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CLINICAL TREATISES
on the
PATHOLOGY *and* THERAPY
of DISORDERS *of*
METABOLISM *and* NUTRITION

By
PROF. DR. CARL VON NOORDEN
Professor of the First Medical Clinic, Vienna

PART IX
TECHNIQUE OF REDUCTION CURES AND GOUT

AUTHORIZED AMERICAN EDITION
Edited and translated under the supervision of
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Chicago, Ill.

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NOTE BY THE AMERICAN EDITOR.

The principles enunciated in the preceding volume of this series relative to the treatment of leanness apply with equal force to the treatment of obesity. In fattening cures the caloric intake should be increased so that it lies above the level of the average diet, whereas in reduction cures the caloric intake should be reduced to such a point that it lies below the ordinary maintenance diet. At the same time individual peculiarities must be carefully studied in each case and the treatment arranged accordingly. Routine treatment or the following of any scheme or schedule is more dangerous here than in the treatment of any other form of metabolic disorder, with the possible exception of diabetes. In the management of obesity the Art of dietetics becomes as important as the Science. In sanatoria and in watering places too much attention is generally bestowed upon the immediate result, too little upon the ultimate effect; the most important element of the treatment being always the education of the patient not for a few weeks, but for the rest of his life. Many of the popular so-called reduction systems that lay claim to be original are dangerous in proportion to their "orig-

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inality" chiefly because they do not take into consideration the comfort and the daily habits and surroundings of the patient, but render it essentially impossible for the patient to carry out the rules laid down for his guidance for any considerable period of time. In this pamphlet these factors are all carefully considered and the arrangement of a régime taught that, while based upon scientific studies of a simple kind, is intensely practical and relatively easy of execution.

In the lecture on Gout and the Uric Acid Diathesis the most modern and the most important aspect discussed relates to the determination of the boundary of tolerance for uric acid forming foods. In the case of diabetes this method has long enjoyed deserved popularity. Just as in diabetes it is generally unnecessary and often dangerous to withdraw all carbohydrate food for an indefinite period of time, so in the uric acid diathesis it will be found equally superfluous and precarious or even practically impossible to completely exclude all uric acid forming food from the diet. In order to determine the boundary of tolerance in sufferers from uric acid disorders the endogenous uric acid excretion must be determined as a preliminary step; and then the degree of exogenous uric acid excretion following the intake of a definite pabulum studied separately and compared with a calculated normal. It will be found that in true uric acid cases definite and clean-cut deviations from this normal become apparent. From the diagnostic standpoint such studies are of the greatest practical importance, especially as the early diagnosis of so-called uric acid lesions generally remains exceedingly uncertain and vague, in as much as most patients are truly gouty before any of the numerous clinical syndromes that are attributable to uric acid perversions make their appearance. If a correct diagnosis can be made early by tolerance determinations, the patients will often be spared weakening and uncomfortable diet restrictions and the imposition of other severe measures that are detrimental rather than otherwise for true sufferers from gout and goutiness.

The therapeutic value of such tolerance determinations is as great in diabetics, for by their aid one can readily determine how much of valuable purin-containing food can be safely given without upsetting the disordered uric acid metabolism and, above all things, without chronically underfeeding the patient. For it will be found that there is generally left some power to properly assimilate purins; and this power should by all means be utilized. The details in regard to the technique of tolerance determinations and the estimation of the endogenous and exogenous uric acid excretion, the tests of purin-free and purin-containing foods, as well as the practical dietetics and the general treatment of the uric acid diathesis, all based upon these studies, are given with great exactness in this volume and make very interesting and instructive reading.

ALFRED C. CROFTAN.

May 9, 1910.
100 State Street, Chicago.

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PART I

TECHNIQUE OF REDUCTION CURES

PART I.

TECHNIQUE OF REDUCTION CURES.

I. GENESIS OF OBESITY.

I will limit myself to a very brief discussion of the factors that lead to obesity. It is necessary, however, to indicate the main rules that should govern the treatment of obesity, for a careful understanding of the underlying principles is necessary before a reduction cure can be intelligently undertaken.

Under normal conditions there is an automatic regulation of the output of energy and the intake of food. A human being after having attained an optimum, that is, a medium average state of nutrition, adjusts himself to a definite amount of food that constitutes for each individual the so-called "maintenance diet"; the latter deviates upward and downward [according to the varying demands created by a varying amount of physical labor performed] from the average medium food intake. Of course, the exact amount of energy that corresponds to the consumption of energy on each day is not introduced during each 24 hours' period; but these differences become so completely equalized by small increases and decreases that many persons for years and for decades maintain their weight, that is, the resultant between intake and output, unchanged.

This finely adjusted normal regulatory mechanism is occasionally disturbed, sometimes forcibly, but more frequently by the cumulation of numerous small causes producing a large effect.

1.—OBESITY FROM OVERFEEDING.

The Consumption of Energy Remains the Same, but the Food Intake Increases.—For one or the other reason, as I will illustrate later on by specific examples, more nourishment than is really necessary is taken. We will assume that the daily plus, that is, the excess above the maintenance diet, amounts on an average to 200 calories. This sum is contained in

1/3 litre of milk
or 25 g of butter
" 200 g fat-free meat
" 145 g moderately fat meat
" 70 g white bread
" 4/10 litres of beer, etc.

After deduction of the incidental expenditures of energy (see Vol. VIII), these additions to the diet in excess of the maintenance diet produce a daily addition of about 20 g. of fat, that is, in a year of 7.3 kg. of fat; and in so far as fat is always deposited in conjunction with other substances, in particular water, an increase in weight (adipose tissue) of about 10 kg. Such slight increases in the intake of energy (only

200 calories per diem) are rarely the result of increasing the *bulk* of the diet; that is, the volume of the daily food intake remains approximately the same, but its composition becomes changed. Let us assume that a bachelor, who has been used to eating in restaurants, marries. At home the food is usually prepared with more fat than in a restaurant; often also more is eaten because more time is spent at meals and because favorite dishes are more frequently served. But, even if this is not the case, the greater amount of fat in the home diet renders it more nutritious and increases its caloric value. Or the habit is formed of drinking a glass of beer with friends before supper. The beer is added to a diet that heretofore constituted a maintenance diet and in an imperceptible but steady manner leads to the ingestion of more calories. If at the end of a long period of time the individual is weighed the effect of this small addition to the daily caloric intake is truly astonishing. Examples of this kind could readily be multiplied, but it is unnecessary to outline all the possibilities. I only wish to call particular attention again to the importance of alcohol. Alcoholic beverages are particularly dangerous on account of their tendency to increase obesity, chiefly because they are not used in the place of other articles of food, but are generally added to a diet that in itself is already sufficient to maintain nutrition. Under these circumstances the complete caloric value of the alcohol becomes utilized as a means of fattening. I have already previously explained that 93 g.

of alcohol protect 70 g. of fat from combustion; and this amount of fat, provided the rest of the diet already attains the level of a maintenance diet, is actually spared and stored each day. Naturally this effect is increased still more when with the surplus alcohol certain other nutritive materials as, for instance, the carbohydrates in beer are introduced. The following beverages represent 93 calories (alcohol plus carbohydrate) and would produce a sparing of 10 g. of fat:

	225	ccm	Berlin Weiss beer
or	180	"	Lager beer
"	200	"	Pilsener beer
"	140	"	Munich beer
"	125	"	Munich Bock beer
"	125	"	English porter
"	145	"	Light Moselle- and Rhine-wine, light Bordeaux
"	75	"	Moderately sweet Champagne
"	65	"	Port wine
"	35	"	Brandy, whisky
"	25	"	Sweet Liqueurs (Chartreuse, Curacao, etc.).

2.—Obesity from Lack of Exercise.

The Intake of Energy Remains the Same, but the Consumption of Energy Decreases.—As indicated above, the desire for food and the actual intake of food under altogether normal conditions correspond to the increase or decrease of energy consumption. Occasionally this natural regulatory process is interrupted; this is seen with particular frequency when individuals, who have been accustomed to taking much exercise, for some reason change their habits and begin to lead a more lazy life. In men following various callings, this frequently occurs towards the fifth decade of life or somewhat later. In women usually a decade earlier, when the children are no longer little and when a part of the domestic duties is assumed by grown-up daughters, housekeepers, and a larger corps of servants. Habits in regard to the amount of food that is eaten remain the same as in past years but less energy is consumed, so that what was formerly a maintenance diet now becomes a surplus diet; and these factors produce, either slowly or rapidly, an increase in the fat deposits of the body. External circumstances of this character are not the only cause that may lead to a more sedate method of life; disease, for instance, may force such a change in habits; I refer to disturbances about the circulatory and respiratory apparatus, or diseases of the nervous system, or of the organs of locomotion. There are innumerable such possibilities and it is very important that the physician should look for any influences of this kind. Very often a little well-directed advice that hits at the core of the difficulty, but at the same time does not too seriously interfere with the ordinary methods of life and the comfort of the patient, may restore the balance between the intake and the output of energy.

I designate all cases of obesity that originate in such a way that the intake of food is only of average size while the output of energy remains below the normal average as "obesity from inactivity." This expression is intended to indicate merely that the musculature is too inactive; it is not intended to cast reflections upon the psychic attitude of these patients, for many of them are by no means deficient in mental energy; they are only prevented by disease, etc., from transforming it into actions.

It is clear that frequently the factors that predispose to "obesity from overfeeding" are combined with factors that predispose to "obesity from lack of exercise," so that the two reinforce one another.

3.—CONSTITUTIONAL OBESITY.—We may consider it as established that in most obese people the amount of energy consumed pro kilo of protoplasm is altogether normal, and that there is no "retardation of the metabolism." Overfeeding alone, that is, a disproportion between the food intake and the energy output, is the cause of the increase in the fat layers. Obese individuals and frequently, too, physicians, when the first regulations directed towards the reduction of obesity fail to produce immediate results, are very much inclined to attribute the obesity and the lack of success in its treatment not to the habits of life of the patients, but rather to some perversion of their metabolism. The deeper, however, one attempts to gain an insight into the peculiarities of each case, the more frequently will one become convinced that it is unnecessary to conjure up unknown factors, for wrong habits of life alone generally suffice to explain the pathogenesis of the obesity. Nevertheless, there are actually cases in which occasionally an exact and conscientious calculation of the diet demonstrates that these patients not only consume an ordinary normal amount of food, but actually eat less than normal, while at the same time their muscular activity is rather above than below the normal average; and, notwithstanding all this, such individuals become obese; so that here one is actually forced to the assumption that less caloric energy is developed per kilogramme of protoplasm than is normal. In such rare instances one may then actually speak of a "retardation of the metabolism" in the old and venerable sense. Experimental investigations in regard to the amount of oxygen consumed in such obese individuals rather bear out this contention.

Formerly I was inclined to look for the cause of this reduced caloric energy development exclusively in a particular inherited or acquired endogenous constitution of the protoplasm. In numerous cases this explanation is probably the correct one. Of recent years, however, attention has been directed to influences emanating from the thyroid gland. The internal secretion of the thyroid to a large extent governs the intensity of the oxydation processes. We know by very exact investigations that in Basedow's disease (hyperthyroidism) the intensity of oxydation processes (the consumption of energy) is increased by 25 to 50%, whereas in myxedema (hypothyroidism) it may be reduced by 20 to 30%. These are extreme conditions, and between the two poles are undoubtedly many intermediary stages. Slight perversions of the internal thyroid function, upward or downward, will hardly stigmatize individuals inflicted in this way as cases of Basedow's disease or of myxedema in the clinical sense. Nevertheless, the energy development in such cases is changed, sometimes more, sometimes less. The ordinary calculations in regard to the maintenance diet are not only upset, but the patients consume more, or less, than an average normal individual would consume under the same external conditions; so that while they are taking the same amount of food as an average individual, they, nevertheless, emaciate or grow obese (see Vol. VIII).*

Hypothyroidism seems to be not infrequently the cause of retarded metabolism. In part a primary insufficiency of the thyroid gland must be incriminated, and in part a secondary insufficiency of an otherwise healthy thyroid, the function of the latter becoming depressed by chemical influences emanating from other glands (so-called chemical correlation). Such remote effects may originate from the following organs: pancreas, sexual glands, hypophysis, suprarenals, thymus. The clinical and experimental experience we have acquired so far justifies this statement. At the same time a great deal of work remains to be done in order to thoroughly clear these problems.

*I refer to my paper on "The Different Forms of Obesity." Medizin. Klinik. 1909. No. 1. Verlag Urban und Schwarzenberg.—for a discussion of the problems touched upon here. I also refer to the experimental investigation of G. von Bergmann, "The Consumption of Matter and Energy in Infantile Myxedema and in General Obesity." Zeitschrift für experim. Pathol. u. Therap. Bd. V. S. 646. 1909.

Considerations of this kind are not only of theoretical interest, but they are of the greatest practical importance. We should always make the attempt to treat etiologically and not only symptomatically. If, therefore, we are confronted with a case of obesity that has developed on the basis of primary or secondary hypofunction of the thyroid, then reduction of the amount of food or increase of the muscular labor would merely constitute symptomatic treatment, whereas the same measures employed in obesity, due to overfeeding or in obesity due to lack of exercise, would possess the dignity of etiologic treatment. In thyreogenous obesity the administration of thyroid preparations more closely approximates an etiologic plan of therapy, and incidentally, as we will have occasion to show, produces fairly good results, whereas the same treatment in the forms of obesity discussed previously and characterized as obesity from overfeeding would be merely symptomatic.

II. INDICATIONS FOR REDUCTION CURES.

I intend to discuss the indications for an obesity cure very briefly, because this subject has been thoroughly discussed in another monograph of this collection (Vol. 1, 1900).

Must a reduction cure be regarded as "weakening"? This question can be definitely answered in the negative, provided an important preliminary inquiry has been properly solved; namely, whether or not there is any indication at all for instituting a reduction cure; and provided one proceeds in the proper manner. The last postulate is of the greatest importance, and I will have occasion to refer to it later in detail. If the indications are properly formulated, and if the proper path is followed, the patient will not become weaker, but on the contrary stronger from the treatment. A reduction cure itself cannot weaken the patients, but mistakes that may be made in instituting a reduction cure can produce this effect.

High degrees of obesity, that is, a body weight exceeding by 25 kg. or more the weight that corresponds to the height of the patient, must almost always be considered an indication for a reduction cure. In children one is accustomed to speak of a high degree of obesity, if the body weight exceeds the average by about 10 kg. If all the organs are healthy, there are no objections to undertaking a reduction cure in such cases. In children, however, and in very old people, even if the organs are sound, a certain amount of care must be exercised. The higher the degree of obesity the slower should one proceed in the latter class of cases.

Medium degrees of obesity (15 to 25 kg. of overweight), occurring in young people and in subjects up to the 6th decade of life and between, may also be considered fit for a reduction cure, provided the individuals are otherwise healthy. Especially in subjects who have to perform a great deal of physical labor, reduction of the fat is exceedingly desirable, because the heart and arteries become overstrained and wear out more rapidly if they have to supply this unnecessary ballast. In old people, however, one should always approach a reduction cure with a great deal of hesitancy, even though one undertakes to reduce their flesh very slowly and under the most careful control of the whole organism, especially of the heart.

Slight degrees of obesity (an overweight of from about 5 to 15 kg.) do not constitute, from the medical standpoint, a cogent reason for undertaking a reduction cure in healthy people, especially if their weight has been constant for a long period of time. In case, however, a tendency to increased weight has become manifest, then one is justified in taking steps to reduce flesh, especially as at this period a loss of weight may be produced by very slight restrictions that can hardly be dignified with the name of a reduction cure. As far as the patients themselves are concerned, the motive that determines them to consult a physician when they are afflicted with mild degrees of obesity is rather vanity than discomfort, and this applies to women, of course, much more commonly than to men. The physician, provided he possesses not only a knowledge of medical practice, but also has an insight into human nature, will hardly underestimate the importance of this factor. If from a false sense of professional dignity he refuses to place his medical knowledge at the disposal of the patient for this purpose, then she will either prescribe a reduction cure for herself or else will entrust the treatment to some quack; in either case, her health is apt to be endangered.

Particular circumstances may render it desirable, even in milder degrees of obesity, that per se do not seem to urgently call for a reduction cure, to proceed actively; for instance, in diseases of the circulatory organs, in chronic bronchitis, pulmonary emphysema, narrowing of the thorax or kyphoskoliosis, chronic diseases of the organs of locomotion, etc. In all these cases it is of value to remove surplus ballast in order to prevent premature wear and tear.

On the other hand, there are a number of conditions that constitute a warning against instituting a reduction cure. Advanced age has already been mentioned. Diabetic subjects as a rule fare better if the adipose tissue remains somewhat above the normal average.

The same applies to cases of genuine arthritis urica. I have frequently seen cases of gout that were essentially harmless and that developed only rare and mild attacks assume a more serious form as soon as an energetic reduction cure was undertaken. It is true that in sufferers from gout the condition of the heart and arteries must be considered, and derangements about these organs may force one to undertake a reduction cure. In all such cases, however, one should proceed with the greatest care and very slowly. And finally, pulmonary tuberculosis must be mentioned. Here, too, a degree of adiposity slightly exceeding the normal is rather beneficial than otherwise, provided the degree of obesity is not too great and does not actually constitute an advanced form. These two groups of cases must be distinguished. In the first group the obesity of tuberculous individuals must be considered as an artificial product of excessive dieting. This should be prevented as indicated in previous paragraphs (see Vol. VIII). Provided, however, this error has once been committed and the patients have become too fat, then the greatest care must be exercised in again reducing the body weight. Such patients tolerate very poorly a reduction in the food intake, and there is always danger of rekindling a latent tuberculous process in the lungs. In the second and smaller group of cases, the patients approximately maintain the same weight that they possessed before the infection occurred, even though they are suffering from fever and are ingesting relatively small quantities of food. I believe that all these latter cases, that so far have only been regarded as medical curiosities, are really afflicted with a perverted thyroid function; and I urgently recommend a careful study of the thyroid in such subjects, both intra vitam and at autopsy.

There is still another disease the progress of which is unfavorably influenced by obesity, namely, contracted kidney; here, therefore, there is a strong indication for instituting a reduction cure, especially in all those patients who are afflicted with an advanced degree of cardiac hypertrophy and who have a tendency to dilatation of the left ventricle. Here again I must advise to proceed with the greatest care and very slowly. There is hardly any other disease in which a rapid reduction cure is more poorly borne than in contracted kidney. The result of a reduction cure in these cases is almost invariably weakness of the whole musculature, including the heart muscle. I am of the opinion that any attempt to institute a reduction cure in contracted kidney should be made in a sanatorium. Incidentally one might mention in this place that occasionally too rapid a reduction cure or a reduction cure that is carried out for too long a time, may initiate chronic renal disease. As a rule, one is here dealing with contracted kidney. My deceased clinical teacher, *C. Gerhardt,* used to call attention to this point in his clinic in every case of contracted kidney that he demonstrated. Later I have seen many cases that corroborated his view.

III. REDUCTION CURES.

From all that has been said it is clear that obesity may be due to a great variety of causes. Before any plan of treatment is formulated the habits of life of the patient must be carefully and exactly determined in order that the cause of the obesity may be elicited in each particular case. This knowledge renders the treatment much more easy and gives greater assurance of ultimate success. A form of obesity, for instance, occurring in an otherwise healthy individual, quite apparently on account of too little physical exercise combined with too much eating, i.e., the ingestion of an amount of food that exceeds average values, should be combated primarily by greater activity and stimulation of all the factors that lead to a consumption of energy; in other words, by an increase of muscular activity. Provided one would limit one's self in a case of this kind to reducing the food intake, one would, of course, obtain some loss in weight, but would at the same time most assuredly do damage to the muscular powers of the individual. On the other hand, there is a class of obese subjects who perform an abundant amount of very strenuous bodily labor, but, nevertheless, grow fat and remain so, because they satisfy the increased demand for food, that is stimulated by this labor, without restraint. In such individuals there is nothing gained by insisting upon a still further increase of muscular activity, for this very measure would only lead to a still greater stimulation of the appetite. Such individuals should be taught to be moderate in eating; in other words, they should be instructed how to satisfy their appetite with articles of food that are filling but possess only a small caloric value.

1.—DIET.—In all cases of progressive obesity the intake of food is greater than the output of energy, and the problem to be solved is to arrange a proper proportion between the two. As soon as this is accomplished the accumulation of fat is arrested. *During the reduction proper, however, the intake of food is to be rendered smaller than the output of energy.* Under such conditions the deficit is borne by the tissues of the body proper. According to the principles that have been formulated in a previous lecture, the body has a tendency to cover this deficit from its adipose tissues, while at the same time manifesting a tendency to spare its albumen content. In obese individuals, who possess an enormous amount of reserve material in their fat layers, the body albumen is protected much better than in subjects who are in a medium or in a poor state of nutrition. Nevertheless, body albumen may be sacrificed if one does not proceed properly. This was shown by the nitrogen balance studied by *F. Hirschfeld,* in 1893. His patients in the course of a reduction cure lost from 1 to

3 g. of nitrogen daily (corresponding to about 6 to 8 g. of albumen). Exactly at the same time *C. Dapper* and I, jointly, published a series of investigations that demonstrated that the loss of nitrogen can be fully obviated and that it is possible to dismiss the patients at the end of a reduction cure with the same amount of body albumen as they possessed when they came. This question has frequently been studied since that time, and I have always kept this point in view. The most important summary of my conclusions is that relatively high amounts of albumen, administered together with relatively high amounts of carbohydrate, are the proper means to protect obese subjects undergoing a reduction cure from loss of nitrogenous body substance. The introduction of fat may and must be correspondingly decreased. I will have occasion to speak of this in more detail later on.

The rules in regard to feeding that obtain in reduction cures must all be formulated upon the science of caloric feeding. This does not imply, of course, that in each case and on each day a tedious calorimetric study of the diet should be carried out by the physician or the patient. Only in really energetic reduction cures (reduction cures of the third degree, see below) do exact caloric determinations become necessary. This does not constitute an impossible task nor one that cannot fairly be demanded, because strict reduction cures after all belong to the domain of sanatorium treatment or should be carried out only by physicians who occupy themselves especially with problems of this character. A physician who is not fully familiar with caloric calculations of the food should never undertake the management of an energetic reduction cure.

It is, of course, impossible in this place to give detailed rules, and I will limit myself to the elucidation of general principles. In every case a general estimate should precede the actual reduction cure in order to determine the true demand of the patient for food (amount of the "maintenance diet"). From the product obtained by the multiplication of the body weight by the caloric demand pro kilo (see Vol. VIII), a certain sum must be deducted in obese subjects, because adipose tissue does not participate in the transformation of energy, this function being relegated exclusively to the protoplasmic substance. One must, therefore, calculate by about how much the body weight of an obese patient exceeds the weight of a normal individual of the same height. This difference must be included in the calculation. We will assume, for instance, that an obese patient weighs 100 kg.

The normal weight of an individual of the same height we will say is 70 kg.

The occupation and the amount of labor performed by the patient we will assume to be such that under normal conditions 37 calories pro kilo of energy would be consumed (see Vol. VIII).

In order to calculate the maintenance diet of such a patient the figure 37 must be multiplied not with his actual weight, that is, 100 kg., but with his normal (ideal) weight, that is, 70 kg. And we arrive thus at the product of 2500 calories as his "maintenance diet." In this method of calculation there are, of course, included certain sources of error, but they are not of great importance. Practically I have found that this method works out very well. If this figure is assumed as the basis, then we must estimate as closely as possible, the nutritive value of the diet that the patient actually is in the habit of ingesting, and we will almost invariably find that this sum is greater than the calculated "maintenance diet." It now becomes a relatively easy matter to determine where one must begin to reduce the diet in order to bring down the total caloric value of the diet that this patient was accustomed to eat; besides, one must determine, in each particular case, whether it is advisable to proceed slowly or fast.

a—The Three Degrees of Reduction Cures.—For practical reasons I distinguish three degrees of reduction diet. I have utilized the above example to illustrate my meaning:

Maintenance diet calculated as 2500 calories.

I. Degree of reduction diet 4/5 of the demand = 2000 calories
II. Degree of reduction diet 3/5 of the demand = 1500 calories
III. Degree of reduction diet 3/5 to 2/5 of the demand = 1500 down to 1000 calories

It is not difficult to arrange Diet I or Diet II. All that is required is to omit all visible fat (butter, oil, meat fat, etc.), to prepare vegetables and dishes made from flour with little fat and to restrict the use of beer and wine; in this way the caloric intake is reduced below the lower boundary of Diet I (in our example 2000 calories). In the case of Diet II dishes made from flour, stewed fruits, milk, and soups containing flour would also have to be forbidden.

Rules of this kind can be carried out at home as well as in sanatoria or health resorts. Their effect is slow, but, provided they are carried out conscientiously, sure. The patients should be instructed, in order to compensate for the lack of the above articles of diet, to eat abundant albuminous pabulum, chiefly lean meat and cheese. Only in cases undoubtedly suffering from arthritis urica or from nephritis should the patients be advised differently.

In Diet III of a reduction cure the bill of fare should consist essentially of the following articles of food:

Coffee, tea without additions,
meat broth (fat skimmed off) with vegetables,
lean meat or fish (total weight 250 to 350 g, weighed cooked),
lean cheese,
abundant green vegetables and salads prepared with as little fat or oil as possible,
vinegar, lemon, vinegar pickles, brine pickles, tomatoes, celery, radishes,
abundant raw fruit, with a small percentage of sugar (apples, peaches, strawberries, raspberries, currants, blueberries, sour cherries, grape fruit, early oranges, etc.),
coarse bread (bran bread, Graham bread) in quantities of from 40 to 70 gm.
potatoes (prepared without fat) in quanties of from 80 to 150 g.,
Mineral waters ad libitum,

Wine, in weak persons, up to 200 gm, preferably omitted altogether,
1 to 2 eggs,
Skimmed milk,
Buttermilk.

With a little experience in practical dietetics a diet can be arranged from the above articles that offers sufficient variety, while at the same time incorporating a very small caloric value. It is, of course, essential to calculate the latter carefully and to adapt it to the requirements of each individual case, as otherwise many unpleasant surprises will be experienced. I must emphasize again that I do not consider it permissible to inaugurate a reduction cure with so great a restriction of the caloric intake (Third Degree of Reduction Diet) in patients who are following their usual occupation. This method must remain limited to institutions. I could demonstrate the truth of this claim by numerous examples that constitute a serious warning.

A few important details relative to the above diet list call for elaboration.

b—Restriction of Fats or Carbohydrates.—The diet calls for almost complete exclusion of fats; and the fat intake can be reduced without difficulty to 30 g. of fat a day. The fat ration cannot very well be reduced below this point, as otherwise articles of food must be excluded that are essential to the maintenance of proper nutrition and to the tasty preparation of the food. Even the leanest meat still contains some fat, and the same applies to eggs, skimmed milk, buttermilk, and lean cheese. Even in bread and in green vegetables a little fat is contained. On the other hand, this diet contains abundant carbohydrate (in fruit, bread, potatoes, buttermilk). During recent years I have rarely gone below 100 g. of carbohydrate a day, and as a rule I permit as much as 120 g.

100 g of carbohydrate are contained in
about 550 g of potatoe
or " 100 g of coarse bread
" " 1000 g of apple
" " 1250 g of strawberries
" " 2000 g of wild raspberries

The chief advantage derived from favoring the carbohydrates is that hereby the body albumen is spared. 120 g. of carbohydrate are much more powerful in this respect than 53 g. of fat, although the latter represent the same caloric value. Another point that is almost equally important is the property of carbohydrate foods to occupy a large volume in proportion to their caloric value (see Vol. VIII) and to be very filling. It is of paramount importance that the appetite of the patients should be satisfied. A reduction cure that leaves the patients hungry is unquestionably a failure. Hunger reduces the general sense of strength and the energy that is required to carry the cure through. The complaint so commonly voiced that the reduction cure makes the patients "nervous" must practically be attributed to vague sensations of hunger. Any patient, moreover, who during the period of the reduction cure suffered continuously from hunger will promptly indulge in dietetic excesses as soon as the reins are loosened. These important principles were correctly appreciated and duly emphasized by *W. Ebstein* a long time ago. He was of the opinion, however, that in order to combat the sensation of hunger the carbohydrates should be greatly reduced and the fats correspondingly increased, as the ingestion of fat food decidedly impaired the appetite. I cannot agree with this. Comparative studies teach me constantly that the desired end is attained more easily if the diet is rendered rich in carbohydrates and poor in fats. There are, of course, also cases in which the reverse order (*Ebstein's* System) is more effective. Most fat people are naturally great fat eaters, and their appetite by no means suffers when the ingestion of fats is increased to the degree permitted by *Ebstein* (about 110 g. pro die). Besides, the benefits accruing to the albumen content of the organism from carbohydrate feeding are sacrificed if the *Ebstein* method is adopted.

It may cause surprise to see potatoes figuring relatively so largely in the diet. Potatoes constitute a valuable article of diet in reduction cures because they combine a large volume with small caloric value. 100 g. correspond to about 95 calories. From the same standpoint certain other bulbous vegetables may be employed in place of potatoes and in order to furnish welcome variety; particularly, however, should fat-free vegetables of this character be chosen, and they should be prepared with little fat:

100 g potatoe	95 calories
100 g onion	50-55 calories
100 g celery roots	55-60 "
100 g carrots	45-50 "
100 g white turnips	40-45 "
100 g young kohlrabi	42-48 "

c—Proteid Ration.—The diet moreover contains relatively abundant albuminous pabulum (meat, eggs, cheese). I consider it advisable to begin with about 120 g. of albumen a day and then gradually to increase the albumen intake to 150 g. or above. More than 180 g. are never required. If we proceed in this way, we know from experience that the body albumens are best protected. This is demonstrated both by my own experiences and those of my pupil *C. Dapper* (Kissingen).

On the basis of the diet that has just been discussed we arrive at the following minimal and maximal values that apply to a reduction diet of degree III:

Minimal			Maximal		
Albumen	120 g	492 cal.	Albumen	180 g	738 cal.
Fat	30 g	280 "	Fat	30 g	280 "
Carbohydrate	100 g	410 "	Carbohydrate	120 g	492 "
		1182 cal.			1510 cal.

d—Water Intake.—So far nothing has been said in regard to the water intake. This question has already been discussed at length in a previous volume of this collection (No. IV). I have for a long time been combating the view originally advanced by *Oertel* and *Schweninger* that the reduction of the water intake increases the oxydation of fat and in this way becomes directly operative as a means to reduce fat; nowadays this opinion is hardly discussed seriously. The consumption of energy is in no way increased by the restriction of water; this is demonstrated by convincing experiments in animals, in healthy and sick individuals. It remains undetermined whether extreme restriction of water, such as one would actually never encounter, might not yield a different result, but from a practical standpoint this is without importance. Recently *E. Heilner* has shown that, per contra, the abundant ingestion of water increases fat catabolism and the transformation of energy in general. This corresponds very well with the experiences in animals recorded by *Henneberg* (Congr. for Internal Med. Wiesbaden, 1885), live stock men having found that the administration of abundant water renders it very difficult to fatten animals. On the other hand it is no doubt true that restriction of the water intake, everything else remaining equal, produces a loss of fat. This effect is indirect. Many people spontaneously suffer a loss of appetite if they are allowed little to drink, especially if the ingestion of liquids is forbidden during meals. In such individuals, therefore, drink restriction constitutes a convenient method of preventing polyphagia and hence incidentally of combating obesity. All people, however, do not react in this way; for some persons eat just as much despite the reduction of liquids, and such people consequently do not become thinner; others, again, suffer a loss of appetite in the beginning, but soon become accustomed to the new régime and again eat more abundantly, so that the initial loss of weight that was warmly welcomed when it occurred, is soon regained. At all events, this simple means of attaining the desired end is worthy of trial in any case. Only in the uric acid diathesis and in cases showing a tendency to stone formation (gallstones!) this measure might turn out to be precarious. Obese diabetics and neurasthenics, also, tolerate the reduction of the liquid intake very badly.

Personally I only employ water restriction if there are complications about the heart and arteries. Here, however, these latter complications and not the obesity become the determining factor. Especially in cases with very high blood pressure is this method of value.[1]

Otherwise I permit free ingestion of liquids in reduction cures in the form of plain drinking water, mineral waters, tea, coffee, fat-free meat broth. The loss of flesh is not impeded hereby, and a great favor is at the same time rendered these patients who are compelled to suffer manifold deprivations in other directions.

e—Measuring Glass and Balance.—In order to properly carry out a reduction cure of the III. degree, often even of the II. degree, measuring glass and balance must be employed. Without these aids errors will constantly creep in on the part of physician and patient so that the success of the whole undertaking may become jeopardized. We are indebted to *Harvey* and his disciple, *Banting,* after whom the oldest system of reduction is named, for having established the principle that food and drink must be weighed and measured. *The introduction of quantitative methods into the regulation of the diet* must be considered a therapeutic achievement of the first importance. Since that time quantitative methods have come into their own in all branches of practical dietetics, and everywhere they have been accompanied by success.

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[1] von Noorden: On Exercise Treatment and Drink Restriction in Circulatory Disorders, Monatschr. f. d. physik.-diät. Heilmethoden. I. Heft 1, 1909.

f—Warning Against Routine Methods.—I have emphasized above that in formulating regulations of a dietetic character intended to counteract obesity no definite schematic plan should be followed. Any one who has familiarized himself with the relations existing between caloric intake and energy output (see Vol. VIII), must understand what an infinitude of modifications may exist in the arrangement of an anti-obesity diet. The essential element always remains the reduction of the caloric intake below the "maintenance diet," and at the same time the introduction of a sufficient quantity of proteids. Within these limits the most manifold modifications are possible. As an example I may quote the so-called milk cure that has recently been recommended. Now, it has been known for a long time that exclusive milk feeding as a rule partakes of the character of a diet restriction, i.e., of a reduction cure.[1] A liter of milk, as a rule, incorporates approximately 625 calories. An obese subject, like the individual illustrated in the example given above, would therefore require four liters of milk in order to maintain nutritive equilibrium. If milk constitutes the only article of diet it becomes very filling. The demand for food in most obese subjects can readily be satisfied by two liters, at most three liters, of milk, and on this diet he must lose flesh, because in the first case he receives only about ½, in the second case only about ¾, of the value of his calculated maintenance diet. Whether it is practical, however, to proceed in this way remains another question. I judge reduction cures, as well as any other systematic method of feeding instituted for the purpose, for instance, of fattening thin subjects, or diabetics, etc., not only from the standpoint of temporary success but also, in particular, from the standpoint of their educational value. The patients should learn, in the course of their treatment, how to arrange their mode of life in the future; hence I have always considered it most expedient to adhere as closely as possible to the ordinary mode of life of each individual and to make changes and restriction in their common schedule appropriate to the particular object in view. If one proceeds in this way the results are apt to be more lasting than if a reduction cure by dieting is undertaken that stands in no relation whatsoever to the patient's ordinary habits nor to his future mode of life. Routine treatment according to a definite scheme or schedule fails properly to teach them the errors that they have committed in the past and that they must avoid in the future. Milk cures have become popular only within the last few months, and, nevertheless, I have already seen four patients who rapidly lost flesh while undergoing this cure, but promptly regained this loss as soon as they became tired of the milk; here a diet invariably had to be decided upon, after all, that more closely simulated their ordinary habits and that was formulated according to old and well-established principles.

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[1] See F. A. Hoffmann, in Leydens Handb.d. Ernährungstherapie, 1897.

What has been said in regard to this milk cure practically applies to other reduction diets that possess some semblance of originality, but that for this very reason are not permanent in their effects. I refer for instance to a strictly vegetarian diet. The latter incorporates abundant carbohydrate, holds little fat and little albumen, and is of a low total caloric value, at least in comparison to its bulk. Preference is given to this diet in *Lahmann's* institute and in other so-called Nature Cure sanatoria. It is, of course, self-evident that a considerable reduction of flesh can be secured by such a diet. While the patients are living under the strict rules of the institution everything proceeds favorably in the beginning; soon, however, the effect of the small albumen ration becomes manifest, and general weakness not infrequently develops later from such a regimen. Besides, patients fed in this way become accustomed to the ingestion of a diet that is altogether too voluminous, so that when they return home they find it difficult to curb their polyphagia, especially if the food either from carelessness or from a desire to render it more palatable is prepared with abundant fat; it is clear that under these circumstances they rapidly regain the flesh they had lost. Many cases, moreover, suffer permanent injury because their musculature grows weak and flabby and they become anaemic as a result of living for so long a time on a diet that is deficient in the proper amount of albuminous pabulum.

Some time ago I had the opportunity of studying the case of a Russian physician who on the basis of theoretic studies had constructed for his own use the following simple reduction diet:

	ALBUMEN	CARBOHYDRATE	FAT		
2 eggs	11.2	0.5	11.0	about	150 cal.
300 g of cheese....	75	10.5	22.0	"	554 "
1000 g of apple.....	3.5	120	0	"	506 "

Total about 1210 cal.

For months this diet, with the addition of water, constituted his sole food; the result obtained was formidable, inasmuch as his weight became reduced from 121 kg. to 79 kg. He consulted me because of late he had noticed a general loss of strength. He did not want to abandon the method of feeding he had adopted, so I advised him to add 4 eggs daily to his diet in order to bring his low albumen intake up to about 110 g. and his total caloric intake up to about

1500. I saw him again several years later, and he expressed his gratitude to me for the advice I had given him, for he soon recovered his strength and permanently retained good general vigor. During all this time he continued to live on the same diet; his body weight finally fell to 72 kg., and, aside from slight fluctuations, thenceforth remained constant. In this case no doubt a "retardation of the metabolism" (see Vol. VIII) occurred, otherwise the scanty number of calories introduced could not have maintained the relatively high body weight of 72 kg. for so long a time. This physician was inclined, when I saw him last, to publish his method of dieting as an effective and commendable reduction cure. I have never been able to determine whether this was actually done. Recently I learned that he fell a victim to cholera last year. If the diet that this patient devised for himself had been a little richer in albumen (an improvement that could easily have been arranged) then no objections could have been formulated against it on general principles. The percentage of carbohydrate and fat corresponds approximately to my own recommendations given above. The loss of flesh must be very considerable. But it is very questionable whether a diet cure as heroic as this one would prove suitable for other patients, and I seriously doubt whether many patients would have the will power or the energy to live on a diet of this character, that closely resembles that of Nomad tribes, for any length of time.

Generally speaking, a diet system will fulfill its object as a reduction cure the less it is "original" and the more it corresponds in its general character to the diet that is generally eaten by each nationality.

2.—Muscular Exercise.—Aside from the arrangement of a suitable diet, the most important task in reduction cures is insistence upon a sufficient amount of muscular exercise. We possess no better, more certain, or more readily controlled means of increasing the output of energy and chiefly the consumption of fat than exercise. In certain cases this element is the most important part of the treatment, namely, in the category that must be included under the group of "Obesity from Inactivity" (see Vol. VIII). One must always remember, however, that it is rarely possible to accomplish the desired purpose in such cases without at the same time regulating the diet; for if the patients are left to their own initiative in regard to the amount of physical exercise they are to perform, the increased appetite and thirst engendered by muscular labor frequently exceed the rise in the oxydative processes produced by the latter. Under such conditions a very desirable strengthening of the musculature is obtained, but at the same time the fat layers of the body also become increased.

In each individual case of obesity the degree of physical exercise and the particular kind of exercise that the patient should perform must be determined. Individuals with a strongly developed muscular system must be ordered to perform much more strenuous physical labor than fat individuals with a weak musculature. In cases of the latter kind, that predominate, of course, in women, but that are not uncommon in men, it is often necessary to begin very moderately, otherwise the fatigue becomes too severe and persists for too long a time, so that the patients become exhausted and lose in energy. Still greater care must, of course, be exercised if the heart is involved. In the latter case it is often necessary to limit physical labor to breathing exercises, for the latter strengthen the heart much more directly than any other form of muscular exertion. It need hardly be emphasized that the most careful daily control on the part of the physician is essential under these conditions.

Whatever the initial amount of physical labor may be, one should always remember that this is an educational process, and one should be quite sure that a given amount of physical exertion can be tolerated without detriment to the patient before proceeding to more strenuous exercises; otherwise the heart may become injured and this injury is apt to remain quite permanent in obese individuals, for here the heart disposes of a smaller amount of reserve force than in a perfectly healthy individual. If all these precautions are carefully observed, muscular exercise will be found to be an exceedingly effective means for reducing flesh. At the same time the danger is obviated of leaving the patient in a weakened condition after the reduction cure is terminated. I have already mentioned in a previous section that there is no more efficient means to produce the optimum of muscular development in an adult than physical exercise (see Vol. VIII). Not only is the retention of nitrogen and albumen favored hereby, but the muscle fibres themselves are increased. An obese individual that attempts to reduce weight merely by undernutrition always incurs the danger of sacrificing body albumen and of reducing the strength of the whole body. If undernutrition, however, is combined with muscular labor the strength of the whole body is rather increased than decreased.

There are many fallacies current relative to the effect of different forms of muscular exercise. The ideal form and, at the same time, the form that can be best regulated is *mountain climbing*. I have already previously shown how one can calculate that an individual weighing 100 kg. rising to an elevation of 1000 m. consumes at least 706 calories (see Vol. VIII). Unfortunately, mountain climbing cannot be carried out everywhere, partially for lack of time and partially on account of geographical conditions. In watering places, that are usually situated in a hilly or mountainous country, mountain climbing, however, constitutes one of the most important factors in the treatment. Walking on the level is only a poor substitute. The amount of energy consumed by this form of walking stands in no proper proportion to the amount of time consumed and to the amount of fatigue that is engendered. A much better means are *sports,* and among these rowing, tennis playing, gymnasium work, swimming occupy first place. Under certain conditions *medical gymnastics* must be utilized, but the significance of this form of exercise should not be overestimated. For the amount of active calorie-consuming labor that is performed during one hour daily with *Zander's,* or *Herz's* apparatus is not very great. Nevertheless, exercise at home may be utilized to supplement other forms of physical labor. I prefer the exercise that *Müller* describes in his book "My System" to all other methods.

Massage exercises no appreciable influence upon the fat metabolism of the individual who is being massaged, but a considerable influence upon the metabolism of the masseur. Neither can *horseback riding* be considered an efficient means for reducing flesh. The passive vibration incident to horseback riding produces only a very mild increase of metabolism in the musculature. The effect of horseback riding upon the bowel action may, of course, be very considerable, and is always welcome, but this advantage is neutralized by the danger of an increased appetite and resulting greater intake of food.

3.—MINERAL WATER CURES.—There are many watering places in which fat people congregate every summer in order to undergo a reduction cure. In Europe Homburg, Carlsbad, Kissingen, Marienbad enjoy the greatest reputation. It is claimed for these waters that they "stimulate metabolism," in other words, increase the general metabolism, in particular, the combustion of fat. This problem is now amenable to exact mensural control. The results obtained are very slight. It is true that within the first and second hour after the intake of mineral waters the output of energy increases by about 5 to 10%, but this is attributable more to the stimulation of the intestinal glands and musculature than to any other factor. Distributed over the whole day this increase in the total output of energy barely exceeds 1%. And not more than 2 to 3 g. of fat are consumed in order to furnish this amount of extra energy, certainly an exceedingly small quantity. In addition, one must remember that the intake of laxative mineral waters sometimes, although not always, somewhat impairs absorption. But not more than 3 to 4 g. of fat are withdrawn from assimilation in this way. Drinking cures are of great value for certain of the complications of obesity, as gastrointestinal disturbances, hepatic derangements, uric acid diathesis, etc. The undesirable reducing effect of such a cure, as it is carried out in the above-mentioned watering places, depends *in its totality* much more upon other factors than upon any specific properties of the waters (diet, exercise, hydrotherapy). Without these accessory factors the drinking cure per se is merely a drop on a hot stone.

4.—HYDROTHERAPY. — Among hydrotherapeutic measures *sweating*, that is, for instance, the Turkish-Irish bath, and steam baths, are most popular as reducing means. Electric sweat baths are also very popular. As a matter of fact very imposing reductions in weight can be attained by these means, but this triumph is very short-lasting. What is actually sacrificed in addition to small quantities of salt and very insignificant quantities of organic products of metabolism, that the kidneys could have gotten rid of much more easily, is water. This water is soon replaced, rapidly if much water is taken, less rapidly if the water intake is at the same time restricted. So much is certain that sweating does not cause a loss of fat. Fat must be starved and worked away. It cannot be sweated away. There is some little justification for sweating procedures in mild degrees of circulatory disturbances, here they aid in removing slight œdemas and in stimulating the peripheral circulation and thereby indirectly also the heart. As soon, however, as one is confronted with circulatory disturbances of a more advanced degree, sweating becomes prohibitive. Arterial disease must also be considered a counterindication.

Carbonated baths are indicated where it appears necessary to exercise a particular influence upon the heart; but they exercise no effect upon the combustion of body fat.

They should never be employed if there is high arterial tension, a phenomenon frequently witnessed in "plethoric" obese men. In such cases I often have recourse to bleeding in the beginning of the treatment (200 to 250 ccm. of blood); this causes a reduction in the blood pressure, and after this preliminary measure carbonated baths are very well borne.

Half baths with a gradual reduction of the temperature of the water, cold rubs, swimming followed by cold douches, are of use not only to stimulate circulatory and respiratory activity, but also to increase the catabolism of fat. These measures withdraw heat that must be replaced by combustion of fat. So far no exact investigations in regard to the degree of caloric output that they produce exist. One can estimate that from 10 to 20 g. of fat must be sacrificed in order to replace the heat lost in a half bath followed by a cold rub. This is a considerable amount of fat. In institutions that treat many cases of obesity *cold-water treatments* are becoming more and more popular; and this is due to the fact that experience has shown them to be effective not only in reducing fat but in hardening the patients while at the same time counteracting the tendency to dangerous bronchitis and disagreeable hyperhydrosis that such individuals are apt to suffer from.

5.—THYROID THERAPY.—I mentioned in a previous chapter that there are cases of obesity due not to eating an extraordinarily abundant diet of a fattening character nor to performing an exceptionally small amount of muscular exercise, but to reduced oxydative energy. As far as we know to-day the cause of this reduction must be looked for in a decrease of the specific function of the thyroid gland. This may be due to a primarily deficient or disordered thyroid function or to the effect of stimuli emanating from other organs and affecting the thyroid function secondarily in such a way that the latter becomes depressed or inhibited. In either case one can speak of *hypothyroidism*. It is by no means easy to correctly interpret conditions of this character. There is only one form in regard to which we have exact knowledge and that we can diagnose without difficulty, namely, the form that is dependent on disease of the hypophysis cerebri (degeneratio adiposo-genitalis). Cases, too, that are associated with myxedema can hardly be misinterpreted (see Vol. VIII).

Frequently, however, the correct state of affairs is not recognized until certain peculiar experiences with reduction cures are made. Patients afflicted in this way may not react favorably to a reduction diet that would otherwise produce the desired effect; increased muscular exercise also produces only slight results. And cases that after the institution of especially rigorous means actually do lose fat and weight, frequently will be found to have become very much weakened and to recover very slowly from the effect of the treatment, while at the same time the loss of weight is rapidly regained. Whenever an experience of this kind is encountered, one should suspect hypothyroidism. Here a thoroughly justified etiologic method of treatment can be advised, namely, the administration of thyroid preparations that act as a bellows for the combustion processes of the organism. I place particular emphasis upon this point, because I have always most energetically opposed the use of thyroid preparation in ordinary obesity due to overfeeding or lack of exercise. In the latter category of cases the benefits accruing from thyroid medication are merely transitory, and it is, of course, impossible to order these patients to eat thyroid substance indefinitely. As soon as the administration of thyroid is stopped the patient's weight increases again, simply because the originally underlying cause of the obesity persists. Such patients do not learn during their treatment how they must live in order to prevent a reformation of fat.

In cases of hypothyroidism it is permissible to administer thyroid preparations permanently or at least for many months at a time with short intermissions. Here the diet should not fall below the maintenance diet, and this is very agreeable to patients who previously were ordered to live on a reduced diet for years and decades. Particularly important is the administration of rather *abundant quantities of albumen,* immaterial whether this albumen is incorporated in meat or in other proteid foods. I agree with *Rheinboldt* in regard to the correctness of the rule that abundant albumen should be administered in this category of cases, even though certain other authors do not occupy this stand. The observations that I have made in my own patients and in patients of other physicians teach me to insist on the administration of large quantities of albumen when thyroid preparations are given. I am not prepared to say that very much depends upon the kind of thyroid preparation that is given. My personal experience is practically limited to *E.*

Merck's Thyreoidinum siccatum, of which 0.1 to 0.3 g. are administered daily. In some of the cases this preparation was given for two years uninterruptedly without producing the slightest disagreeable effects. I have abandoned the use of iodothyrin, that I formerly employed quite extensively, because it did not seem to be sufficiently effective in the customary doses (about 1.0 g. daily). As soon as I administered larger quantities (2.0 to 3.0 gm.) that exercised a definite effect upon the body weight, I would sooner or later hear complaints in regard to considerable irritation of the heart. The dose of any thyroid preparation is to be regulated in such a way that not more than 500 to 800 g. of body weight are sacrificed in one week.

PART II

GOUT, NEPHROLITHIASIS URICA AND THEIR TREATMENT

PART II.

GOUT, NEPHROLITHIASIS URICA AND THEIR TREATMENT.

I. DEFINITION OF GOUT.

In the case of *gout* one is confronted with difficulties in attempting to formulate any exact definition of the disease. We know that in this disorder the fate of uric acid differs from the normal, in so far as deposits of urates occur in the body, and certain disturbances in the excretion of uric acid make their appearance. We know, further, that in the vicinity of urate deposits inflammatory processes are apt to develop that run either a chronic or an acute febrile course; furthermore that the tissues that have become permeated with urates (especially cartilage) very readily undergo degeneration and necrosis, in this way causing serious interference with the function of the affected joints. Beyond this we know very little that is positive in regard to the significance of uric acid in the production of gouty processes. We do not even know whether the formation and destruction of uric acid deviates quantitatively from the normal. It is clear that the visible outward evidences of gout (urate deposits and articular inflammations) are merely the *consequences of gouty disease,* and an individual is afflicted with gout long before such visible evidence of gout appears. There are no doubt many cases in which metabolic perversions characteristic of gout are present and in which numerous consequences of these perversions appear, without the occurrence at the same time of specific gouty deposits. Unfortunately we know so little in regard to these stages of gout that precede the period of visible manifestations that we can hardly diagnose them with certainty. Only within recent times have certain endeavors been made to secure a solid basis for this diagnosis. Unfortunately numerous vague symptoms of varying character, as fleeting pains in the extremities, in the body, the head, attacks of migraine, gastrointestinal disturbances are often christened with the name "Gout" simply because no other diagnosis can be made. As a rule, this diagnosis is merely the result of ignorance and it can rarely tolerate the light of scientific criticism.

In the course of this lecture I will also have occasion to speak of the internal treatment of ***nephrolithiasis urica,*** because the treatment of this disease corresponds in many respects with the treatment of gout. At the same time it is questionable whether the deposit of urate concretions in the renal pelvis really has anything to do with the gouty process. We are probably dealing with two diseases that are fundamentally different in the following respects, namely, that in gout the retention of uric acid in the organism plays an important rôle, whereas in nephrolithiasis urica uric acid is excreted in normal (perhaps even supranormal) quantities but is subsequently deposited in a crystalline form *outside of the body tissues proper* on account of an unfavorable composition of the liquids that are expected to hold it in solution. In studying the nature of the two diseases one encounters more points of difference than of correspondence; nevertheless, in both disorders the same cause may increase or decrease the morbid process and this justifies the joint discussion of the two disorders, at least from a therapeutic standpoint; for in both cases anything that leads to an increase in the formation of uric acid must be considered detrimental.

Unfortunately, the definition of the term *"gout"* is exceedingly confused and uncertain. I have already mentioned the fact that numerous diseases associated with pain are unreservedly declared to be gout, among them *neuralgia, myalgia, diseases of the tendon sheaths,* of the *periosteum,* of the *bones.* If in such cases an analysis of the urine reveals high absolute or (as is usually the case) only high percentic values for uric acid, then the diagnosis of gout (see below) is often made. Very serious diagnostic errors in this direction are of common occurrence. The great majority of symptoms that are grouped under the name of gout are really due to rheumatic disease. They are attributable to masked forms of acute articular rheumatism and like the latter can be cured in a short time by the prompt administration of sufficiently large doses of salicylic acid and antipyrin. Frequently luetic disorders of the muscles and periosteum lead to confusion. Even unquestionably rheumatic neuralgias and myaglias may be diagnosed as gout. If no further error were committed than a wrong diagnosis, the patients would not be particularly injured, but unfortunately the diagnosis of gout immediately conjures up an army of dietetic restrictions and other far-reaching measures that are by no means indifferent in their effect upon the general strength of the patient, while at the same time the best period for proper treatment is sacrificed. A *rheumatic neuralgia* that could have been cured within a week by the daily administration of from 5 to 6 g. of sodium salicylate may drag along for weeks and even months, if these patients are treated by an antigouty diet, by exhausting baths and diet, by massage that is apt to increase rather than decrease inflammatory disorders. Frequently one is told that a neuralgia must have been gouty as salicyclic acid did not help. As soon as a more careful inquiry is made, however, it will be found that only from 1 to 3 g. of aspirin or of some similar preparation were given, and that merely for a few days. Such small doses never suffice to cure acute inflammatory rheumatism. I see each month several cases of *luetic myositis* or *periostitis* that have been mistreated for months by weakening baths, mineral water cures, restriction of the food and a weakening diet, simply because the disease was considered to be gout. One could elaborate almost indefinitely on this subject.

It is equally deplorable that the nature of certain articular lesions is very frequently misinterpreted. *Acute inflammatory rheumatism* is relatively rarely diagnosed as gout, although here the possibility of error is greatest. Mistakes are more frequently made in the opposite direction, in so far as genuine gouty disorders are often misinterpreted as acute rheumatic polyarthritis. *A. Magnus-Levy* some time ago called attention to the fact that acute polyarthritis occurring *for the first time* in an individual over 50 years old is always very suspicious of gout. With this statement I fully agree. This suspicion is strengthened if only a single joint or merely two or three joints are affected and when the characteristic jumping from joint to joint so often seen in acute rheumatism is absent.

Still more frequently *chronic articular disorders* are falsely considered to be gout. Here the monoarticular and oligoarticular forms of *true articular rheumatism* that run a slow course from the beginning and that are by no means rare must be considered (especially when affecting the knee joints), also *tabetic* and less frequently *tuberculous joint lesions*. The latter, if the disease runs an atypical course (and this is more common in older individuals than in young people) may so closely simulate the symptom complex of genuine gouty articular lesions that the correct diagnosis may be exceedingly difficult and can frequently not be arrived at until an operation is performed. Even X-ray pictures occasionally fail to settle the question. Particularly important in this respect are the atrophic degenerative joint lesions that are usually included under the name of *arthritis deformans.* To this group belong *malum coxae senile* and corresponding changes about the shoulder joints and the vertebra. These are often falsely called gouty. This error is still more frequently committed in the case of deformities that develop about the phalanges of the fingers. Occasionally the changes are limited to thickening of the joint capsule edges producing the well-known protuberances known under the name of *"Heberden's Nodes";* in other cases there develop advanced wear and destruction of the whole joint capsule and of the head of the phalanx. These degenerative processes are originally not of an inflammatory character, the lesions should therefore more appropriately be designated "Arthrosis" rather than "Arthritis." Secondarily, however, inflammatory changes may appear in the adjacent soft parts causing considerable pain. All of these diseases have nothing to do with arthritis urica, for the diseased joints are completely free from uric acid. It may, of course, occur that an individual suffering from one of these forms of degenerative arthrosis becomes at the same time affected with genuine gout; and in such a case the gouty process is easily engrafted upon joints that are already diseased. If, therefore, in isolated cases urate concretions have been found in such joints, this merely proves that gout complicated the original disorder without indicating that the primary process was in itself gouty. To consider Heberden's disease as evidence of the existence of uric acid diathesis is exceedingly careless diagnostic work.

Genuine gout is common in some regions and rare in others. In Austria-Hungary the disease is very rare. During the 2½ years that I have been working in Vienna I have seen only 25 cases of genuine arthritis urica, notwithstanding the fact that I am justified in assuming that gouty patients are somewhat concentrated in my particular practice. Of these 25 gouty cases 20 were foreigners. In Frankfort during the same period of time I saw twice as many gouty cases recruited from the region of middle Rhine, the Palatine, and the Mosel region. The frequency with which the diagnosis "gout" is made in Austria is in marked contrast to the actual frequency of genuine arthritis urica. This is attributable to the fact that Heberden's disease, which is very common, is always christened with the name of gout.

II. ON THE PLACE OCCUPIED BY URIC ACID IN THE METABOLISM OF GOUT.

In regard to the ultimate cause of gouty disease and the manner in which the fate of uric acid is determined thereby, we know essentially nothing. For the present we are limited in our studies of gout to deliberations relative to the metabolism of uric acid. It will be found that in gouty subjects as a rule symptoms of *uric acid retention* make their appearance, although this phenomenon does not always become manifest throughout the whole long course of the disease.

1.—PRELIMINARY CONSIDERATIONS.—In addition to uric acid, its congeners the xanthine bases that accompany it wherever it appears in the body must be discussed. Both together, that is, uric acid plus xanthine bases, are included under the name of alloxuric bodies or better purin bodies. At one time it appeared that one of the peculiarities of gout was a change in the normal quantitative relation existing between uric acid and the xanthin bases. This view, however, was based upon inexact analytic methods and soon had to be abandoned. From a practical standpoint xanthin bases are unimportant. One should know, however, that the uric acid that is eliminated from the body is invariably accompanied by xanthin bases, the latter constituting from 10 to 12% of the excreted uric acid by weight. It is also important to know that the xanthin bases and uric acid both originate from the same source.

2.—SOURCES OF URIC ACID.—Only those substances that contain the purin nucleus discovered by *E. Fischer* can be considered sources of uric acid. From this purin nucleus are derived in addition to uric acid the bases adenin, hypoxanthin, guanin, and xanthin. The purin nucleus is a derivative of nucleinic acid that, in combination with proteid radicles, forms nucleoproteids. As purin-containing nucleoproteids occur almost exclusively in the nuclei of animal cells, this nuclear substance must be considered the most important source of the purin bodies of the urine. In addition, a certain amount of hypoxanthin found in muscles and no doubt derived from the nuclein of the muscle nuclei must be considered. It has never been definitely established whether or not uric acid is formed synthetically in the mammalian organism *from other bodies than nucleinic acid.*

We must distinguish:

1. Uric acid formed within the body itself and without the introduction of uric acid forming pabulum: *endogenous uric acid.*

2. Uric acid derived from the nucleinic acid and the purin bases of the food: *exogenous uric acid.*

a—Endogenous Uric Acid.—Even when uric acid forming pabulum is completely excluded from the diet the organism continues to excrete uric acid (and purin bases) because a continuous disintegration of cell nuclei occurs and because some of the hypoxanthin of the muscles is continuously eliminated and must be replaced from the nuclei of the muscle cells. During inanition this wasted amount is presumably not replaced, hence when the food supply is cut off the organism is exceedingly sparing with its store of nuclein (see first lecture, Vol. VIII). If nuclein-free pabulum, however, is ingested the organism promptly replaces the wasted purin material. For the present we do not know at all from which elements this synthesis occurs. So much is known that the body is capable of reconstructing the purin nucleus from material that does not contain the latter. The infantile organism possesses this power to a very great extent, for the nuclein substance of the infantile body increases rapidly even though milk contains only traces of purin material.

I also consider it probable that the adult organism constructs its nuclein *exclusively from purin-free material* and that this is due to the power inherent in the nuclear substance to construct the purin nucleus. I consider it, furthermore, exceedingly doubtful whether any of the purin bases of meat or any of the nucleoproteids ingested contribute in any way to the building up of the body nucleins.

The endogenous uric acid excretion is determined by administering a purin-free diet, for instance, milk, eggs, numerous fruits, and vegetables (see below).

Under these circumstances we find from 0.36 to 0.5 g. of uric acid in the daily urine. It is true that several days have to elapse before such low values are obtained and before the latter remain on a constant level.

EXAMPLE:

I.	Day of purin-free diet...........	0.71 g uric acid
II.	" " " "	0.65 g " "
III.	" " " "	0.52 g " "
IV.	" " " "	0.42 g " "
V.	" " " "	0.43 g " "

This endogenous value (in this instance 0.42 to 0.43 g.) can hardly be influenced, at least in a healthy subject. It must be considered a biologic constant of the individual. At the same time a few factors are known that can exercise at least a transitory effect upon the height of this excretion, some of them increasing, some of them decreasing, it:

Salicyic acid and its derivatives, mineral acids, thymic acid, possibly also saline mineral waters increase the values for endogenous uric acid a little, while alcohol decreases them. In the first instance we are probably dealing with better elimination, in the second instance with a certain amount of *retention* of uric acid. It is very improbable that any of these substances exercise any influence upon the *formation* of uric acid; it is possible, however, that alcohol occupies a peculiar position in this respect (see below). At the expiration of a few days the original values for the endogenous uric acid excretion seem to become reestablished, even though some of the above substances are being administered.

Under pathologic conditions the values for endogenous uric acid can increase considerably. This is the case when abundant nuclear substance undergoes degeneration. One finds, for instance, very frequently

In a variety of febrile diseases	0.7—0.8 g	uric	acid
" pneumonia	0.7—1.2 g	"	"
" advanced stages of leukemia	0.7—2.0 g	"	"
" leukemics at the beginning of X-ray treatment	0.7—2.5 g	"	"
" progressive severe diabetes	0.7—0.9 g	"	"

It is worthy of mention in this place that there are certain individuals who, although perfectly healthy, excrete large quantities of endogenous uric acid. In the case of a perfectly healthy man of 45 years, who was repeatedly examined in this respect in the course of the last 10 years, we never found values for the endogenous uric acid below 0.7 to 0.8 g. A very obese lady of about 35, who lived for 2 weeks exclusively on 2 liters of milk and 500 g. of apples per diem (reduction cure) excreted in the beginning of the treatment from 1.1 to 1.4 g. of uric acid. At the end of the 2 weeks' milk-apple period it was found that the uric acid excretion had only fallen to 0.85 to 0.9 g. This patient was afflicted with no disorder other than obesity. This observation was recorded in the year 1904 and the patient has remained perfectly well in the meantime. I will have occasion to mention below that in cases of nephrolithiasis very high endogenous uric acid values are occasionally observed.

In genuine gout the value for endogenous uric acid is, as a rule, reduced (0.28 to 0.4 g.), excepting in the acute stages (see below).

b—Exogenous Uric Acid.—As soon as purin-containing food is introduced into the body the excretion of uric acid increases. The bases contained in the diet (above all, quantitatively predominating, the hypoxanthin of meat) are presumably absorbed without undergoing any preliminary change. A portion is destroyed and transformed into urea; another portion is excreted unchanged or in the form of uric acid. It remains undetermined whether the bases that we ingest become an integral part of the organism (hypoxanthin of muscles or components of nuclein, see above). Nuclein is split in the intestinal canal into its component parts, namely, proteid and nucleinic acid. A portion of the nucleinic acid is sacrificed to the activity of intestinal bacteria. The bodies of the intestinal bacteria, together with disintegration products of desquamating intestinal epithelia furnish the purin bodies that are recovered in the feces. Another portion of the nucleinic acid is absorbed and presumably disassimilated to a great extent in the intestinal wall. Participating in the latter activity is a particular ferment, *nuclease,* that produces this splitting off of purin bases from nucleinic acid in the intestinal walls or in any other part of the body where this process occurs.

Inasmuch as a portion of the purin bodies of the diet escapes absorption altogether, whereas another portion is disassimilated to a point where urea is the end product, it is clear that the whole of the introduced material does not appear in the urine as uric acid (or purin bases). On an average 50% disappears, whereas the other half is recovered in the urine.

The values for the exogenous uric acid excretion, therefore, in contradistinction to the endogenous values and very fluctuating. They are generally dependent upon the quantity and the quality of the meat consumed. Beef on an average contains so much nuclein-and hypoxanthin-material that 0.18 g. of uric acid can be generated therefrom. Of this only one half appears in the urine. With a daily consumption of 100, 300, 500 g. of beef, the following uric acid excretion may be expected in an individual whose endogenous uric acid value amounts to 0.5 g.:

Uric acid	100 g meat	300 g meat	500 g meat
endogenous	0.5 g	0.5 g	0.5 g
exogenous	0.09 g	0.27 g	0.45 g
Total	0.59 g	0.77 g	0.95 g

All these considerations lead to a fundamental axiom, viz.: that from urinalysis alone one can never conclude whether or not the uric acid excretion of a patient is normal, decreased, or increased. The amount of purin containing material that is ingested must always be compared with the output of uric acid. It is important to know rather the balance between these two factors than the absolute figures for the uric acid excretion.

3.—Uric Acid Factors in Gout.—Our positive knowledge in regard to the metabolism of uric acid in gout is very scanty. I will not enter into a detailed discussion of this subject but will limit myself to a statement of the most important established facts.

1. The value for the endogenous uric acid excretion is, as a rule, reduced or remains near the lower level of the normal. Between the attacks from 0.28 to 0.4 g. of uric acid are found.

2. The uric acid of the blood is increased and it is very difficult to rid the blood of uric acid even though a purin-free diet is administered for a long period of time. The average amount of uric acid found in a liter of blood taken from a gouty subject was 70 mg.; normally only traces being found.

3. Urate deposits in joints and tendons as well as muscular and cutaneous tophi are often found. The tophi are not of constant size. They may grow larger and smaller. The reduction in the size of tophi is essentially due to phagocytosis inasmuch as the urate needles are devoured by the cells. In the joints (cartilage, epiphysis, ligaments, etc.) the disappearance of urate deposits seems to be much more difficult and to proceed much more slowly than in the skin and in the muscles.

4. The excretion of exogenous uric acid is insufficient and retarded. Frequently only one-half, one-third, or still less of the uric acid that one would ex-

pect to be eliminated according to the diet reappears in the urine.

5. The acute attack of gout is spontaneously accompanied by a greatly increased excretion of uric acid, in other words, the organism becomes purified of uric acid during the attack.

All in all, therefore, one finds in a gouty subject a chronic retention of uric acid, interrupted by an occasional critical elimination associated closely with more or less violent local inflammatory phenomena and general toxic symptoms.

I will refrain from discussing the theoretical conclusions that may be drawn from these fully established facts and from a series of other less positive and less constant observations of a different kind. I refer for an elucidation of this subject to my Handbook on the Pathology of Metabolism (1907).

The material that is positive renders it possible.

1. To strengthen the diagnosis of gout:

2. To determine the tolerance of gouty subjects for purin bodies and to regulate the treatment accordingly.

a—The Diagnostic Significance of Uric Acid Elimination.—In all those diseases that can be confounded with arthritis urica the elimination of endogenous uric acid is not so low as in chronic gout. There is one exception to this rule, namely, very old age. Here the metabolism of the nuclear substance is apparently reduced, and hence old people eliminate from 20 to 25% less uric acid on a purin-free diet than vigorous adult individuals. This point must, of course, always be considered in each case of gout occurring in an old person. I may add that the latter react to the introduction of purin containing material in the same way and with the same increase of the uric acid elimination as younger persons. In many articular disorders, especially in acute articular rheumatism, very high values for the endogenous uric acid may even be found (*v. Herwerden,* Zeitschr. f. klin. Med., 65. 19. 1908).

In non-gouty articular lesions, especially in chronic articular rheumatism and in arthrosis deformans, the purin bodies of the food reappear in the urine almost as promptly as in a healthy individual. It is true, I have occasionally seen a retardation of the elimination by one day; but on the following days the expected excretion of uric acid took place. In one case in which it was necessary to decide whether a chronic and painful swelling of the knee joint was of gouty origin or not we found the following values:

Endogenous uric acid values on the 4th to the 6th day of the purin-free diet:

0.46 g
0.42 g
0.45 g

Average 0.45 g

On the next 2 days the patient received 400 g. of beef (weighed raw), and then again for 4 days a purin-free diet; theoretically the ingestion of 800 g.

of meat should have caused an elimination of 0.72 of uric acid. There were found:

0.49
0.97
0.56
0.44
0.53
0.43
Total 3.42

The endogenous uric acid excretion during the 6 days of observation should have been 2.7 g. (6 times 0.45 g.); as a matter of fact 0.72 g. more were eliminated, that is, exactly the calculated sum. So exact a correspondence will, of course, rarely be discovered. As a rule the reclaimed quantities of uric acid fluctuate by 10 to 15% upwards or downwards from the calculated sum. The prompt elimination of uric acid decidedly militated against the diagnosis of gout. Later a dense fibrous pseudoankylosis of the joint developed. This never occurs in arthritis urica, and hence definitely indicated (in the same sense as the metabolic study that had been performed half a year before) that the case belonged to the group of chronic articular forms of rheumatism.

In nephrolithiasis uratica the elimination of uric acid, after the administration of purin containing food, follows the ordinary rule. I find that in this disease the patients develop very high values for endogenous uric acid. In three cases, for instance, I was unsuccessful in reducing the uric acid elimination below 0.65, 0.72, 1.02, notwithstanding the fact that the patients lived on a strict milk diet for a whole week. In a number of other cases I noticed a low uric acid production, but the values for the uric acid always remained near the upper boundary of the normal. This seems to demonstrate that in the pathogenesis of uratic renal concretions an unusually high formation of uric acid may play a rôle, added, of course, to unfavorable conditions of solubility in the urine (high salt percentage, excess of acid phosphates). This may be due to the fact that these individuals are afflicted with increased nucleolysis or that a relatively large portion of the destroyed nucleinic acid is transformed into uric acid and only a relatively small portion to urea. Only such cases, of course, were considered in which the fresh urine contained no uric acid of older origin in the sediment.

In gout the results of examinations of this kind yield altogether different results, at least in certain stages of the disease. During the acute attack, as already mentioned, there occurs a spontaneous and very considerable increase of the urinary uric acid. It seems logical to conclude that at this time uric acid is washed out of urate deposits in joints, tophi, etc. Studies in regard to the production of exogenous uric acid during this time have not been made with sufficient thoroughness, and this problem is difficult to solve because one cannot decide how much of the uric acid increase is due to liberation of uric acid from urate deposits and how much to exogenous factors.

During the intervals between typical gouty attacks there are no doubt long periods during which the endogenous and the exogenous uric acid production is altogether normal, or, at least, varies so slightly from the normal that the deviations cannot be recognized. On the other hand, anomalous conditions may occur that do not correspond with the facts commonly observed in chronic atypical gout. Investigations in regard to the elimination of exogenous uric acid are, therefore, of diagnostic value in these studies only if they give a positive result, that is, if a considerable retardation in the uric acid elimination can be determined.

This constitutes no particular disadvantage as far as the diagnosis is concerned because typical cases of gout interrupted by free intervals offer no particular diagnostic difficulties, and because there will usually be found a tophus here and there that facilitates the diagnosis. In one case a positive result from this test proved to be of great value. The patient was a man who had never suffered from a typical attack of gout but had a typical gouty heredity (grandfather, father, and older brother). The patient consulted me in Frankfort en route to Homburg (1906), where he had been sent on account of fleeting neuralgiaform pains. These pains had been interpreted as symptoms precursory to gout, and, as was shown later, correctly. A metabolic study revealed great retention of exogenous uric acid, only a third of the expected quantity being found in the urine. This pointed to the diagnosis gout. About two weeks later, during the course of his cure in Homburg, the patient suffered his first typical attack of podagra.

In atypical chronic gout in which the local manifestations run an exceedingly slow course and in which periods altogether free from any disturbances do not occur or are exceedingly short, a retention of exogenous uric acid is almost invariably present. The endogenous uric acid excretion also deviates from the normal in so far as the values for the latter are usually exceptionally low, and in so far as greater fluctuations in the daily elimination are observed than in a healthy subject. If now to the purin-free diet purin containing material is added for two days the urinary elimination of uric acid increases only very slowly; or a short attempt at active elimination occurs that soon, however, becomes abortive. Even if the patient is placed for a whole week upon a purin-free diet after the expiration of the two meat days, only about one-half of the calculated amount of uric acid may reappear in the urine. This phenomenon is so constant that in doubtful cases it must be credited with great diagnostic importance. Therapeutically, too, it is valuable in so far as it indicates whether or not the patient should be troubled with the bothersome, occasionally dangerous and weakening restriction of purin containing foods.

I will now quote a few examples:

Day	Diet	Chronic articular rheumatism	Chronic atypical gout
1	purin-free	0.65	0.49
2	"	0.52	0.52
3	"	0.425	0.34
4	"	0.45	0.36
5	addition of 400 g beef......	0.61	0.39
6	purin-free	0.60	0.41
7	"	0.49	0.40
8	"	0.40	0.46

The 400 g. of beef should cause an elimination in the urine of 0.35 g. of uric acid over and above the endogenous value for uric acid. In the rheumatic individual this endogenous value (average of 3rd and 4th day) was 0.44 g. The increased elimination of the 5th and 6th day calculated together amounted to 0.33 g. The whole calculated quantity, therefore, appeared in the urine at the proper time.

In the gouty individual the endogenous uric acid value was 0.35 g. (average of the 4th and 5th days). The addition of meat produced a very much retarded increase in the uric acid elimination. Within 4 days only 0.26 g. instead of the expected 0.36 g. were eliminated (together 1.40 g., instead of 1.66 g.).

b—The Therapeutic Significance of Tolerance Determinations.—Three years and a half ago I called attention to the therapeutic importance of tolerance determinations in gouty subjects in a little monograph that I published together with my former assistant, *L. Schliep* (Berl. klin. Wochenschrift, 1905. No. 41). Even though we do not understand the exact nature of the gouty process, still all the facts that we are acquainted with to-day, as well as clinical experience, indicate that everything must be done in this disorder to prevent the retention of uric acid. One can take the stand that the best treatment is to exclude all purin containing material from the diet and this requirement would be filled by the use of vegetables, milk, and eggs (see below). Articles of this kind, provided they are given in sufficient quantity and in correct combinations supply a sufficient amount of adequately nourishing material. As a matter of fact, it is altogether rational in severe cases of the disease, especially in very painful forms of chronic arthritis urica to adopt this diet and to continue its use for weeks or even months at a time. The results obtained are frequently surprisingly good. I can remember a considerable number of cases in which the patients, while they did not get rid of the deformities that had occurred, nevertheless were spared all further difficulties, even though for years they had never been free from pain and had always been afflicted with new swellings. In some patients it is even necessary to maintain permanently a milk-egg-vegetable diet, because even the slightest addition of purin containing material to the food is not well tolerated and invariably precipitates new attacks.

Fortunately, however, only a small proportion of sufferers from gout is so completely intolerant to purin containing pabulum. In chronic atypical gout, as well as in the free intervals of typical gout there always remains a certain power of transforming and eliminating a limited quantity of purin material in a normal manner. The amount that can be utilized in this way corresponds to the *boundary of tolerance.* Danger does not accrue to these patients until this latter is exceeded, consequently it is important to know the boundary of tolerance. Conditions are here exactly similar as in diabetes mellitus. In the latter disease we have learned that it is forbidden and dangerous to adopt the routine of feeding every diabetic upon the same diet. We have learned to recognize that a most exact study of the tolerance of diabetics for carbohydrates must precede the regulation of the diet and that the latter must be adapted to individual peculiarities; for example, one patient will excrete sugar on 50 g. of bread, whereas another individual can tolerate 150 g. of bread without glycosuria; we find similar differences in the case of gouty subjects as far as their tolerance for purin containing material is concerned. Gout and diabetes, moreover, are related in this respect that the tolerance does not remain constant, but may fluctuate in either direction; such patients occasionally tolerating more and at other times less purin containing material. An improvement in the tolerance may be attained therapeutically if the intake of purins is considerably reduced for a certain length of time, because in this way the uric acid forming function is, one may say, spared or rested, and an opportunity is given for its recuperation. It has also occasionally been observed that small quantities of purin containing material may increase the excretion of uric acid more, *relatively,* than large quantities, and may even produce an *absolutely* higher uric acid excretion. Quite analogous conditions are found in diabetes.

An example of this kind is mentioned in the monograph quoted above (*von Noorden* and *Schliep*); another one is herewith submitted. The diet in this case had been purin-free for five days preceding this investigation, but the urine was not analyzed:

Day	Diet	Uric acid
1	purin-free	0.33
2	"	0.41
3	"	0.29
4	"	0.34
5	addition of 400 g beef	0.38
6	the same	0.53
7	purin-free	0.41
8	"	0.57
9	"	0.36
10	"	0.36
11-20	purin-free	Not determined
21	purin-free	0.37
22	"	0.28
23	"	0.37
24	addition of 200 g beef	0.49
25	the same	0.55
26	purin-free	0.62

The endogenous uric acid excretion of this patient, who was afflicted with chronic atypical gout amounted to 0.34 g. (average of the 1st to 4th day). The addition of 800 g. of beef (400 g. on the 5th and 6th days) should have produced a plus of 0.72 of uric acid in the urine. As a matter of fact, from the 5th to the 8th day only an excess of 0.52 g. above the endogenous value appeared, then the excretion became reduced. From the figures of the 21st to the 23rd day an endogenous average value of 0.34 g. could again be calculated. The addition of 400 g. of beef (200 g. for 2 days) should have produced 0.36 g. of uric acid, whereas the excess excretion of the 24th to the 26th day amounted to 0.64 g. The addition, therefore, of a smaller amount of beef not only relatively increased the uric acid elimination, but rendered it absolutely higher than the administration of the larger quantity of beef.

I need hardly call particular attention to the fact that (in contradistinction to commonly accepted views) the elimination of quantities of uric acid that are large in proportion to the amount of purin containing food ingested indicates a favorable condition of affairs. For a gouty individual is only then placed in a dangerous position when uric acid is retained in the body.

III. THE DIETETICS OF GOUT AND NEPHROLITHIASIS URICA.

Just as the physician who prescribes a diet for a diabetic should know the carbohydrate percentage of the different foods that he allows, so any one who undertakes to treat gout rationally should know the purin percentage of the food administered. In the latter case it is true matters are very much simpler than in the case of the carbohydrate content of the diet.

1.—PURIN-FREE FOODS.—The products of female mammals and birds, that is, milk and eggs, must be considered purin-free. Traces of purin bodies, it is true, are found even in these articles of food, but from a practical standpoint they are negligible. It is immaterial whether the milk is administered in the natural state or fermented (sour milk, Yaourt); the derivatives of milk: cheese, butter, cream, milk sugar, casein, and casein preparations are, of course, also free from purin bodies. In the case of cheese certain reservations must be made, in so far as cheese, on account of the large quantities of bacteria that it harbors, may incorporate considerable quantities of purin bodies. Fish eggs (caviar) are also permitted in gout. An analysis made by *S. Fränkel* (Vienna) showed: water 55.2%, nitrogen 5.1%, no trace of purin bodies.

All vegetables, with a few exceptions, contain so little purin that they can be used freely by gouty subjects. Most foods of vegetable origin are altogether purin-free, as, for instance, all varieties of sugar, vegetable oils, flour, leaves, roots, and stalks. In seeds, on the other hand, purin bodies have been found, but they are limited altogether to the plant embryos contained in seeds, and the latter occupy only a small part of the kernel. Nevertheless, it might be advisable in particularly severe cases of gout to forbid the use of seeds altogether (peas, lentils, hulled beans, maize, wheat, etc.), whereas, there is no objection to the use of flour made from these different seeds, for in process of preparation the purin containing plant embryo is completely removed from the flour.

Coffee beans, tea leaves, cocoa beans must be particularly mentioned because they contain abundant purin. Caffein is transformed into heteroxanthin, theobromin into methylxanthin, theophyllin into paraxanthin. In view of these transformations these bodies have very little to do with the formation of uric acid for the above purin bases are in no way related to gout in man. Moderate quantities of coffee, tea, and cocoa are, therefore, permitted without reservation; the use of larger quantities is, however, to be condemned on the basis of practical experience.

2.—Purin-containing Foods.—Aside from the exceptions enumerated above, animal tissues alone need be considered as purin containing substances that lead to the production of exogenous uric acid. In the tissues the purin bodies are either present as such (hypoxanthin of the muscles) or they are split off from nuclein in process of digestion and absorption (see above). Tissues containing the largest number of cells are generally richest in nucleins, hence the glandular organs in which the cells are crowded closely together like thymus, pancreas, liver, spleen occupy first place. It is, hence, altogether rational both in gout and in nephrolithiasis to forbid altogether the use of *internal organs*. There is one exception to this rule, namely, the liver of fattened fowl (fat liver of geese) because there is relatively much protoplasm, full of fat, and comparatively little nuclear substance. The pieces of liver in Strassburg paté de foi gras of the first quality contain less purins than any kind of meat with the exception of fish meat. Among the varieties of meat proper (muscle meat) the *flesh of young animals* contains considerably more nuclein than that of older animals. This is due to the fact that the former contains more nuclei relatively per unit of protoplasm. This factor, however, is somewhat neutralized by the fact that the flesh of older muscular animals incorporates more hypoxanthin. The meat of fish, as a rule, contains less nuclei than that of mammals and birds, and also contains less hypoxanthin. However, fish is not very filling, hence a much larger quantity is usually eaten than of other varieties of meat; consequently if an unlimited amount

Veal

of fish is allowed, probably as much of purin bodies is ingested as in the case of any other meat.

There has been much discussion in regard to differences said to exist between *white and dark meats.* For about 50 years the view has been gaining adherents, both among physicians and the laity, that there are such fundamental differences between these two varieties of meat that subjects afflicted with gout, nephrolithiasis, nephritis, etc., may eat white meat without reservation, but should carefully avoid dark meats. I have been opposing this idea for a long time, in fact, ever since my pupils, *Th. Offer* and *Rosenquist,* demonstrated that the purin content of light and of dark fresh meat is essentially the same. The flesh of different individuals of the same species; the flesh of young and old animals of the same species; even pieces of meat taken from different parts of the body of the same animal showed greater differences in the percentage of nuclein and purin than were found on an average between chicken on the one hand and beef on the other. These facts, it is true, have been denied by certain authors, for instance, *Senator;* but, nevertheless, they are true and irrefragable. The careful analyses of *J. Walker Hall* also corroborate our old findings. Recently *Th. Offer* has resumed these investigations with more careful methods that are altogether above criticism. This thesis has not yet been published, but *Offer* has permitted me to see his protocols, and they fully corroborate what I have said above. There are also a few experiments on record that I made with some of my pupils, and others performed by *J. Walker Hall,* in which meat of different species incorporating the same total amount of nitrogen was fed with the result that the uric acid production was the same whether white or dark meat was administered. All this is fully strengthened by clinical observations that I have now been carrying on for 10 years. I have learned to omit from my regulations any orders relative to distinctions between white and dark, and limit myself to regulating the total amount of meat allowed; and I must declare myself altogether satisfied with the results obtained. One should not underestimate the importance of this step from the practical standpoint; for it is very convenient in treating these cases, not to have to separate the two kinds of meat, particularly veal and chicken on the one side, mutton and beef on the other; for many patients tire excessively of white meat if it is given daily, so that they not only eat less meat than they should, but also suffer from lack of appetite with resulting serious detriment to the general nutrition. It is high time that the old prejudice against dark meat should be abandoned and the true facts of the case be recognized.

Of course, the method of preparation is of the greatest influence upon the purin content of meat dishes. When meat is roasted, and especially when it is grilled, a hard crust immediately forms on the outside that effectually prevents an outflow of the meat juices; consequently all the nuclein and hypoxanthin of the meat is retained throughout its mass with the exception of the outermost layers. When meat is boiled or stewed, on the other hand, a large proportion, and possibly the whole quantity of the soluble xanthin bases are extracted. The latter substances are then found in the soup (meat broth) and in the gravy. When it is prepared as soup, meat loses so much of its purins, by the long process of boiling, that it compares favorably, as far as its purin content is concerned, with fish, that, as stated above, contains the smallest amount of purins of any flesh. Meat broths, gravies, and meat extracts, on the other hand, are forbidden. In pickling (ham, etc.) and in making corned beef the greater part of the soluble purin bases is extracted, whereas the nuclein, of course, remains behind.

The following is a table reproduced from *J. Walker Hall.* It relates to the amount of purin nitrogen (nuclein purin[1] bases purin) contained in raw meat.

Species	Purin nitrogen[1]
Calves' thymus	0.4025%
Calves liver	0.1078 -0.1125%
Beef steak	0.0826%
Loin of beef	0.0506 -0.0538%
Chicken	0.0495 -0.0546%
Turkey	0.0504%
Ham	0.0505%
Loin of veal	0.0454 -0.0481%
Loin of pork	0.0485%

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[1] The method employed by Walker Hall yields results that are somewhat below the correct value. For a table of comparisons, however, this is without significance, as all the figures are influenced in the same direction.

Ribs of beef	0.0455%	
Salmon	0.0450	-0.0482%
Mutton	0.0411%	
Halibut	0.0405%	
Rabbit	0.0305	-0.0456%
Sole	0.0334%	
Codfish	0.0219	-0.0233%

On the lower boundary, therefore, will be found certain varieties of fish, at the upper boundary (if we exclude calves' thymus and calves' liver that are absolutely forbidden) completely fat-free beef that is utilized for beef steak. Otherwise we find that white and dark meats are jumbled together. In particular, chicken under certain circumstances shows even higher values than ordinary beef used for roasting. Ribs of beef, that are very popular in the kitchen, occupy the same plane as the white meat of veal or pork. From the practical standpoint all the varieties of meat that stand between beef and salmon in the table may be considered equivalent as far as their purin content is concerned.

These data by no means exhaust the number of good analyses that we possess. The newer analyses, by *Offer,* that exceed all others that have been published in exactness, disclose facts that might militate in many ways against white meat (especially chicken). It would be very important to possessick more numerous determinations of the purin content of different varieties of meat, for upon such determinations should be based the dietetic prescriptions that we should give patients suffering from gout or from nephrolithiasis.

The *basic diet* must always remain the purin-free foods. As an *accessory diet* to this a certain amount of purin containing material may be added according to the peculiarities of each individual case. The amount of this purin containing material may approach the zero point (see Vol. VIII), or it may, on the other hand, amount to considerable quantities. All this is settled by *determinations of the boundary of tolerance.* The following articles of food must be considered about equivalent as far as their effect upon the increase of uric acid excretion is concerned.

200 g white fish,
100 g ordinary beef, veal, pork, mutton, chicken, turkey, pheasant, venison, ham, salmon, salmon trout,
70 g of beef steak, freed from all fat and connective tissues.

3.—CONCERNING ALCOHOL.—I am no foe of alcohol, and I consider the use of moderate quantities of alcoholic beverages not only permitted, but often even useful. In patients, however, that have a tendency to uric acid retention (gout) and to an increased production of uric acid alcohol in any form or quantity is an evil. It can be demonstrated in most cases that alcohol retards the elimination of uric acid. If the individual is placed upon a perfectly constant diet, so that the values for the uric acid excretion reach a definite and constant level, then it will be found that the use of alcohol invariably produces a reduction in the uric acid excretion. This has been tried with 25 g. of alcohol a day, certainly not an excessive quantity. In a healthy individual this preliminary retention of uric acid is followed by a compensatory increase in the uric acid excretion, and occasionally even a higher level of the urinary uric acid excretion is reached and maintained for a considerable time, indicating that the production of uric acid has been increased.[1]

This increase, however, amounts to only a few centigrammes a day, a minimal amount as compared to the increase in the uric acid production that 100 g. of meat added to the diet can produce. A healthy individual, in other words, need not fear an increase of this kind, for the organism can without difficulty get rid of much greater quantities of uric acid. A gouty individual, however, is seriously affected even by this minimal increase, particularly when it is combined with retention of uric acid induced by the same cause. The same applies to nephrolithiasis, a disease in which we should also carefully avoid everything that can increase the production of uric acid. It remains undetermined whether the kind of alcoholic liquor that is taken makes much difference, careful investigations in regard to this point still being lacking. Clinical experience seems to show that the use of strong spirituous liquors or of heavy wines (Port, Sherry, etc.) and of beer are most detrimental. Light white wines

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[1] The experiments of *Chittenden* also seem to demonstrate that alcohol can increase the production of uric acid, at least temporarily.

(Mosel[1] and light Rhine wines, on the other hand, that recently have unjustly been much neglected), on the other hand, seem to be much less harmful. Comparative studies in this field are urgently needed.

In this place it is important to mention that according to the investigations of *Bloch* and *Pollak,* that have been corroborated in my clinic, chronic drinkers occasionally develop a retardation of the exogenous uric acid elimination similar to that seen in patients with genuine arthritis urica. It is true that here the same degree of retardation is not found, also that this phenomenon is limited to individuals who are excessive users of alcohol. This point must always be considered in utilizing the purin tolerance for diagnostic purposes (see above). The abuse of alcohol no doubt plays a most important rôle in the etiology of gout, although frequently it is more the alcoholism of preceding generations than of the patient himself that is to blame. It is undecided whether or not in such alcoholics, who show a retardation of the uric acid elimination, there is not present as a complication the specific perversion of the metabolism that is always found in gout, even though visible manifestations of gout had not yet become apparent (masked gout). Compare above, page .

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[1] I cannot refrain from opposing the common prejudice that light Mosel- and Saar-wines are to be permitted without reservation in gout, obesity and hepatic disorders, whereas the light Rhine wines are to be forbidden. My personal experience does not coincide with this view. Mosel-wines contain a little more of free vegetable acids and are usually prepared with more carbonic acid in order to satisfy the demands of the modern palate; otherwise there are no fundamental differences between them and Rhine wines. If one wants to pay small or a medium price (1½ to 2½ marks per bottle) better and finer Rhine- than Mosel-wines (incorporating the same amount of alcohol) can be secured.

Patients with gout and nephrolithiasis should be urgently advised to use alcohol-free fruit juices as a table beverage in place of wine; the former can be made from grapes, or from apples, raspberries, currants, etc. This recalls the well-known and ever highly prized grape cure for gout. These beverages grant the patient a drink that is very nourishing and completely purin-free.

4.—COMPOSITION OF THE DIET.—In diabetes mellitus I proposed to divide the diet into two groups, and this plan has been almost universally adopted.

1. *Main diet,* carbohydrate free.—No restrictions in regard to the quantity; that is, the amount taken should correspond to the state of nutrition of the patient.

2. *Accessory diet,* containing carbohydrates.—The quantity is to be adapted by the physician exactly to the individual peculiarities of each case.

In an exactly similar manner the diet in patients suffering from gout or nephrolithiasis may be divided into two groups:

1. *Main diet, purin-free;* no restrictions in regard to the amount excepting as determined by the general state of nutrition of the patient. Care should be taken above all things that a sufficient amount of albumen is incorporated in the diet.

2. *Accessory diet, containing purins.* The quantity is to be adapted by the physician to the individual peculiarities of each case.

As has been stated above (Vol. VIII) the following articles must be included under the category of a purin-free "main diet":

1. Milk and all preparations made from milk; (care in the case of cheese that contains many bacteria or fungi).

2. Eggs of all kinds, also caviar.

3. All vegetables that are at all considered in the ordinary bill of fare, and, above all, also all varieties of flour and sugar made from plants. In particularly severe cases, care should be taken in using the seeds of plants, as well as coffee, tea, and cocoa. I should like to call particular attention in this place to caffein-free coffee (trade mark "Hag") that has a very satisfying taste and incorporates only very slight quantities of caffein.

So-called spices that contain irritating materials as mustard, pepper, paprica, curry, leek, garlic, horseradish, radishes, parsley, etc., are especially to be avoided on account of the danger of irritating the kidneys, for one should never forget that in arthritis urica as well as in nephrolithiasis urica there is always a certain predisposition to renal disease. The above-named vegetable products, however, have nothing to do with the formation of uric acid.

Of acids vinegar is, as a rule, forbidden, while lemon juice is permitted. This distinction is, however, not justified. The small quantities of vinegar that are necessary to prepare a dish of salad can do no harm, for vinegar stands in no relation to the formation of uric acid and does not irritate the kidneys as such at all, because it is immediately oxydized to carbonic acid and water in the body. Citric acid, however, is oxydized with more difficulty.

A suitable arrangement of a *"Main Diet"* would be the following:

Breakfast: Milk, or coffee with milk, milk-cocoa or chocolate.
Porridge made from oatmeal, Grape Nuts, or hominy, or green corn.
Bread with butter, or honey, or jelly.
Eggs in different form.
Fresh fruit, for instance, peaches, baked apple with cream.

Luncheon: Dishes made from eggs.
Green vegetables or salad.
Potatoes or sweet potatoes with butter.
Rice, macaroni, green corn, etc.
Bread with butter and cheese.
Fruits, nuts, almonds, figs, dates.
Milk, sweet or sour cream.
Grape fruit, ice cream, water ice, lemon-squash.
Coffee and cream.

Tea: Tea with cream, cakes with butter or jam.

Supper: Soup made from cereals, legumes, potatoes, or soup made from bone or bone marrow with different vegetables, as tomatoes, cauliflower, artichokes, etc., and cream and parmesan cheese.

Eggs prepared with milk, cream, butter, cheese. Vegetables of various kinds.

Dishes made from flour, potatoes, rice, noodles, macaroni, etc.; milk, cream, eggs, butter, oil, etc., may be used in the preparation of these dishes.

Abundant vegetables of different kinds, with the exceptions of vegetables containing irritating ingredients. Particular attention should be paid to an abundant vegetable diet presenting plenty of variety. Salads made from different vegetables with oil and vinegar or lemon.

Bread and other bakery goods, fresh cheese.

Fruit raw, or boiled or dried, in abundance.

Desserts of all kinds, especially those made from fruit or fruit juices.

Milk, sour milk, curdled milk, cream.

As beverages: fruit juice, unfermented, made from grapes, apples, or raspberries.

2. *Accessory diet.*—A certain amount of meat or fish is permitted as an addition to the above diet, a great deal depending upon the individual peculiarities of each case. The *unit, quantitatively,* is 100 g. of roast beef (weighed roasted). The following quanti-

ties of other meats and fish are equivalent to this as far as their uric acid forming capacity is concerned:

100 g. roast meat (beef, veal, lean pork, mutton, warm or cold ham, ox tongue, calves tongue, venison, rabbit).

100 g. of salmon, salmon trout.

70 g. beef steak, veal cutlet.

140 g. boiled meat (beef, veal, mutton, pork).

200 g. of fish (with the exception of salmon or salmon trout).

200 g. of lobster, crab.

2 dozen oysters.

2 pigeons, or one spring chicken, or ½ a capon of average size, or 1 guinea hen, or ½ a duck, or ¼ of a goose.

The physician may, according to varying circumstances, order 1 or 1½ or 2 portions of this accessory diet, rarely more; in other cases, he will have to forbid it altogether or exclude one or the other variety of meat on account of certain morbid conditions present. As a rule the patient should be allowed free choice among the articles of diet enumerated above.

A very good plan, originally suggested by *Umber*, is to forbid on one day of each week all the articles of diet that are included under the category of additional diet, even though they are permitted on the other days of the week in certain quantities.

The amount of alcoholic beverages that is permitted should, of course, be carefully prescribed by the physician.

It may appear rather complicated to adopt the method described above of separating the purin-free articles of food designated as the "Main Diet" from the purin containing articles designated as "Additional Diet" in such a rigid manner, and to demand that the patient should use weights and measures in using the latter.

But the method is very valuable and has proven of such great use in actual practice that these objections become invalid. In the beginning, of course, the patients must measure their tolerance, but they soon learn without careful measuring how much of the "Additional Diet" they can take.

This is the only method by which the physician can exercise real control over the quantity of uric acid forming material that a patient ingests.

5.—MINERAL WATERS AND DRUGS.—Whereas, heretofore gout and nephrolithiasis could be discussed from a uniform standpoint, this cannot be done in discussing the use of mineral waters and drugs in these two disorders. For by means of these remedies we aim to achieve perfectly different things in the two diseases.

a—Gout.—In the case of gout we must decide whether or not there are means that can counteract the retention of uric acid. One will naturally favor any measure that can produce, however slight, an acceleration of the uric acid elimination. Formerly alkaline waters and alkaline remedies (soda, lithia, magnesia) were very popular, and no doubt alkaline drinking cures often help in gout. Most sufferers from gout, aside from being afflicted with difficulties in the elimination of uric acid and with gouty deposits, also suffer from other disturbances as, for instance, gastric and intestinal dyspepsia of different kind, enlargement of the liver, gallstones, etc., all frequent complications that are favorably influenced by alkaline drinking cures. Hence these patients at the termination of the cure find themselves materially benefited. As far as the uric acid metabolism is concerned such improvement does not, however, become manifest. The idea that the excretion of uric acid can be increased and the body thereby rid of surplus uric acid by alkaline waters cannot be maintained. I merely call attention to the old experience that sufferers from gout are frequently surprised by a typical attack of podagra while they are undergoing a drinking cure in Carlsbad. It appears that this is brought about by a preliminary retention of uric acid resulting from the ingestion of the alkalie. The overloading of the organism with uric acid then precipitates an attack, and this event signals the beginning of the improvement, for during the attack proper the elimination of uric acid increases materially. This interpretation that is borne out by modern clinical and experimental investigations corresponds throughout with the old interpretation given most brilliantly by *Sir Alfred Garrod.* In healthy individuals suffering from typical gout the disagreeable features accompanying an acute attack need hardly be feared, especially as a long period of well-being is very apt to follow such an attack, and, in fact, further attacks may be definitely prevented thereby. There is no objection, therefore, to ordering an alkaline drinking cure from time to time for patients suffering from typical gout. Carlsbad, Marienbad, Neuenahr, Vichy, etc., will always maintain their old reputation in this regard. On the other hand, I urgently warn against administering small quantities of alkalie *permanently* to sufferers from gout. All that is accomplished thereby is an interruption of the normal undisturbed elimination of uric acid. For many years I have forbidden my gouty patients the daily use of alkaline table waters (Fachinger, Obersalzbrunnen, Vichy-Celestin, etc.), and I believe that I can justify this interdiction by favorable experiences obtained in actual practice. *F. A. Falkenstein* decidedly deserves credit for having introduced the hydrochloric acid treatment in typical gout in place of the old alkaline treatment. I emphasize this particularly because I cannot agree with the theoretical basis of *Falkenstein's* hydrochloric acid treatment of gout. (See my Handbook of the Pathology of Metabolism, Vol. II, page 145; footnote, 1907.) *Falkenstein* argues that the administration of hydrochloric acid favors the transformation of uric acid to urea and oxalic acid. If this were the case hydrochloric acid would, of course, reduce the elimination of uric acid, but this does not occur. In a gouty subject one is more apt to witness the opposite; namely, an increase in the elimination of uric acid. It is possible that the administration of large quantities of strong hydrochloric acid loosens uric acid from its connections, hence mobolizes it and improves the conditions that favor its elimination. Alkalies, on the other hand, as the above investigations corroborate, act rather unfavorably than otherwise upon the elimination of uric acid in a gouty subject.

Whereas, in typical gout for reasons mentioned above an alkaline drinking cure may occasionally be decided upon, this does not apply to cases of chronic atypical gout (permanent forms of gouty articular disease). In a paper published some 13 years ago I called attention to the fact that empirically better results are obtained from the use of saline mineral waters in atypical gout than from alkaline mineral waters. Experience had shown that the waters of Homburg were especially effective, and so for decades many patients from England sought this resort with good results. In Germany, where practical experience with typical gout is much more limited, the significance of saline mineral waters has not been appreciated to this extent. I find that the opinion that I expressed at that time has since been reinforced by a very much more abundant experience. The first publication on this subject (On the Influence of Weak Salines on Metabolism. Frankfort, 1896. Published by *J. Alt*) was followed by clinical and experimental investigations reported in full jointly with *C. Dapper* in my Handbook on the Pathology of Metabolism, Vol. II, page 569, ff. Saline mineral waters, in fact, particularly the waters of Homburg and also the waters of the Rakoczy Spring, in Kissingen, were tested, and all of them showed both in healthy individuals and in sufferers from gout a very striking influence upon the elimination of uric acid. It is possible with the aid of these waters to cause the excretion of astonishing quantities of uric acid. The old empiric doctrine, therefore, coincides fully with the facts elicited by clinical and experimental investigations; although an explanation of the method by which better mobilization of uric acid is brought about is still forthcoming.

b.—Nephrolithiasis Urica.—In the case of uratic renal concretions conditions are altogether different. Here an endeavor must be made to reduce the production of uric acid to a minimum, as in arthritis urica, and in addition to increase the solubility of uric acid in the urine to a maximum. This is particularly important in these patients as against cases suffering with gout, because the latter are predisposed to an abnormally large production of uric acid (see Vol. VIII). One of the most valuable means we possess to produce better solubility of urinary uric acid is an abundance of liquids, because the latter render the urine dilute; in addition a certain amount of alkalie is necessary; for, while alkalies rather solidify than dissolve acid sodium urate as it occurs in tophi, they act as a powerful solvent for free uric acid. It is doubtful, of course, whether it is possible to dissolve the dense granules of a larger concretion consisting of a consolidated conglomerate of free uric acid in the renal pelvis, but it is probable, nevertheless, that a certain amount of loosening and disintegration of larger conglomerates to smaller ones can be produced. So much is certain that uric acid, that has not yet formed such solid conglomerations, can be prevented from precipitating by changes brought about in the reaction of the urine. In other words, further concretions can be prevented, although it is not certain by any means that concretions already formed can be redissolved. It is hence very important to counteract too great degrees of acidity of the urine. Hence nephrolithiasis will always be a fruitful field for alkalie therapy carried out by the continuous administration of small quantities of alkaline salts, of alkaline waters, or by a sojourn at some watering place with alkaline springs.

It is, of course, necessary to prevent the urine from becoming alkaline; for it is well known that earthy phosphates are very apt to precipitate upon uric acid concretions as soon as the reaction of the urine turns alkaline. In my earliest publications on this subject I called attention to the advisability of studying the individual acid curve of the urine as a preliminary step and to regulate the quantity and the administration of alkaline remedies on this basis. (Ueber die Beeinflussung der Harnreaktion. Münchener mediz. Wochenschrift. 1888. Nr. 39.) I have frequently had occasion later to note that this is the correct way of proceeding and a very important preliminary step, for I frequently saw patients seriously injured by the routine administration of alkalies without such careful initial studies. In the meantime, my attention was called to the importance of calcium carbonate in the treatment of uratic concretions. The investigations of *A. Ritter* and others had shown that the power of the urine to hold uric acid in solution was essentially dependent upon the relative proportion of mono-sodium phosphate and di-sodium phosphate present in the urine. The former precipitates uric acid; the latter readily dissolves it. If calcium is administered a large portion of the phosphates contained in the diet is fixed in the form of insoluble calcium phosphate in the intestine, another portion is assimilated but is reëxcreted into the bowel via the intestinal mucosa in combination with calcium. This leads to a great decrease of the total phosphates in the urine and, as has been shown experimentally, of the mono-sodium phosphate that is particularly detrimental. Even though very formidable quantities of calcium are administered, an alkaline reaction is never produced in the urine. (Von Noorden, Zur Behandlung der harnsauren Nierenkonkremente. Kongress für innere Medizin. Wiesbaden, 1896. p. 308:—also the publications of my pupils, *J. Strauss,* Zeitschr. für klin. Med. Vol. 31, p. 492. 1897, and *G. Herxheimer,* Berl. klin. Wochenschr. 1897. Nr. 29.) Calcium carbonate is, therefore, an almost ideal method of prescribing alkalies in nephrolithiasis. This corresponds altogether with old empiric findings; for the waters that enjoy the greatest reputation in the treatment of nephrolithiasis are the waters of the Helen Spring, in Wildungen, Fachinger water, the waters of Contrexeville, and of recent years the waters of the Driburg Caspar Heinrich Spring, the Rudolf Spring, in Marienbad, the waters of Rohitsch. All these waters are distinguished by a high percentage of calcium (lime).

Of remedies that have been recommended urotropin acts in the same sense, that is, as a solvent for free uric acid. Nucleinic acid seems to possess the same property. Inasmuch as urea is capable of counteracting the precipitation of uric acid, nucleinic acid and urea have been united in a chemical compound that has been brought on the market under the name of Urol. All of these preparations, however, are far inferior in their effect upon the solubility of uric acid to calcium carbonate.

Whereas, we are capable by medicinal means of contributing a little to the solubility of uric acid concretions in the urinary passages, we are less able to exercise this effect upon the elimination of uric acid in genuine arthritis urica. The old established colchicum preparations seem to exercise such an effect, at least during the time of the attack, and to accelerate the critical outpouring of uric acid that accompanies gouty seizures. At other times than during the attacks and in the free intervals of typical gout and in chronic atypical gout colchicum is altogether inert. It certainly seems worth while to study the effects of colchicum more carefully than heretofore. A remedy that has become so prominent purely empirically merits much more careful attention and study than has been given it in the publications of the last decades. Of salicylic acid and its derivatives, one can say positively that they favor the elimination of uric acid in gouty subjects, but it is a precarious matter to employ them for a long period of time. Salicylic acid is by no means without effect upon the kidneys, and the latter are always in danger in gout. Of recent years thymic acid, a derivative of nucleinic acid, is frequently found mentioned. It is said of this remedy that it materially favors the elimination of uric acid; the drug is known under the name of Solurol (daily dose 0.75 to 1.5 g.). So far our experience with this remedy is limited, and it is too early to draw any definite conclusions in regard to its efficacy. As far as we know to-day it is not practical to continue the use of Solurol for long periods of time, while, on the other hand, a course of treatment with Solurol, lasting about a week and repeated with interruptions from time to time, seems capable of causing the elimination of considerable quantities of retained uric acid.

6.—REMARKS ON LEANNESS AND OBESITY IN GOUT.—Gout occurs both in individuals with abundant and *with scanty adipose tissue.* In the latter group gout is decidely less frequent; it is particularly that form of gout which is produced by chronic lead intoxication that is encountered in thin people. Here the form in which gout appears is usually very severe and involvement of the kidneys as a rule occurs very early. In lead workers, afflicted with gout, whose general nutrition is greatly reduced, I frequently try to improve their general strength by a fattening diet. I have never, however, been satisfied with the results obtained. It is not a difficult matter to increase their weight, but as soon as this is accomplished a disproportion very readily develops between the powers of the heart and the mass of the body, so that cardiac difficulties make their appearance and the danger of cardiac insufficiency must always be feared. I, therefore, advise against any attempt to fatten subjects afflicted with gout, especially with lead gout.

Arthritis urica much more commonly accompanies an *obese constitution,* hence the venerable but by no means correct idea that gout is a specific sequel of high living. A certain connection no doubt exists between gout and obesity. So far, however, we are not able to analyze this relation scientifically and are limited to the teachings of experience. Just as emphatically as I advised against any attempt to fatten a lean sufferer from gout, so emphatically do I recommend attempting to reduce the fat as early as possible in any obese sufferer from gout. If this is not done the heart becomes injured. I need hardly call attention to the fact that even in simple obesity insufficiency of the heart muscle is an extraordinarily common phenomenon. In gout certain additional noxious factors affect the heart; for we must assume that in this disease certain toxic substances (endogenous intoxication, autointoxication), not necessarily, however, uric acid, exercise this poisonous effect. It is very doubtful whether the most careful diet can prevent the peculiar self-intoxication that occurs in a gouty organism. The one thing we can do is to keep the production of uric acid at the lowest possible level and in this way suppress the severe exacerbations of the gouty process. A gouty individual, however, remains gouty even though no more attacks occur and no more uric acid tophi are deposited. Even after typical manifestations of gout (attacks, tophi) have been suppressed, disagreeable and even dangerous sequelae very frequently develop, consisting in cloudiness of the lense, iritis, sciatica, neuritis, also dangerous neuritis of the vagus nerve, nephritis. The nature of these disturbances seems to indicate that certain toxic factors are operative in producing them. Among the organs that are most frequently affected by these unknown, but in my opinion decidedly real, toxic factors, is the heart muscle itself. In a case, therefore, in which gout and obesity are combined, the heart is threatened from two directions (toxic injury from gout, mechanical injury from obesity). One of these dangers we can remove by early interference, that is, by producing a gradual loss of weight by means of careful restriction of the diet. No doubt many of the most brilliant results obtained in regard to the general well-being and the general strength of gouty subjects in Carlsbad, Homburg, Kissingen, Marienbad, are attributable primarily to the loss of weight that is accomplished. On the other hand, numerous failures, in particular injury to the heart muscle, are chronicled in watering places, particularly in Carlsbad and Marienbad. The latter occur at once, or more frequently some time after, a reduction cure has been undertaken. Physicians in general, therefore, have become strongly prejudiced within recent years against reduction cures in gout and thoroughly condemn them. I am not altogether in accord with this prejudice, for the dangers of a reduction cure can certainly be obviated if active steps are taken in early stages, that is, in medium degrees of obesity, or if, at least, every effort is put forward to prevent any further increase in weight. In more advanced degrees of obesity and even in cases in which the combination of gout and obesity has already produced the first signs of cardiac insufficiency the urgent indication to reduce flesh can also be met, provided only that one proceeds with sufficient care. I have carried out many such reduction cures in obese individuals with a weak heart, but only under the most careful control in a hospital or a sanatorium, and I have never witnessed any unfavorable effects upon the heart. On the contrary, a great improvement and a permanent one about the heart is obtained from this treatment in all such patients.

In this place I must insert a very important observation. In the preceding paragraphs I took the stand that sufferers from gout should be permanently placed upon a diet poor in purins, and I gave instructions how this is best done. If it is desired, however, to institute an energetic reduction cure in a sufferer from gout one should deviate from this plan. When I first began to occupy myself with studies relating to the pathology and the treatment of metabolic diseases, I occupied another standpoint and attempted to combine a reduction cure with the administration of a purin-free diet. Thus, for instance, a diet incorporating about 1500 to 1700 calories was arranged, consisting largely of vegetables with the addition of a little egg and milk. Later, however, I abandoned this diet altogether because I saw cardiac complications develop in several instances, and I then decided to adopt a diet incorporating abundant albumen and one especially rich in meat; and it is only since that time that I have secured permanent and favorable results from reduction cures instituted in gouty subjects. In arranging such a reduction cure, therefore, the perversion of the uric acid metabolism should temporarily be relegated to the background to be reconsidered later when the loss of weight has been accomplished. True, I occasionally had the misfortune of seeing a typical attack of gout develop during the reduction cure. These attacks, however, were of short duration; and the ultimate effects, as far as the general health and strength of the patients, and in particular the condition of the heart were concerned, were excellent.

I place particular emphasis upon these studies and experiences because there is in modern therapeutics a tendency toward the same errors that I committed formerly and that I have since recognized and learned to overcome. In many watering places where obese sufferers from gout congregate, a vegetable régime is instituted. Whatever one's views may be in regard to the justification of a vegetarian mode of life, one cannot conceal the fact that the combination of such a régime with a weakening drinking cure, with undernutrition and a great amount of muscular exercise, combined with exhausting hydrotherapeutic means, etc., as instituted in Carlsbad, includes certain elements of danger for any individual, but particularly for a person not used to a vegetarian diet. I can formulate the same criticism against milk cures and vegetarian reduction cures, that have recently become so popular and that are given in many sanatoria, for instance, in Lahmann's institute for the treatment of gouty sufferers from obesity. During the cure proper everything proceeds splendidly, and for a short time afterwards the patients feel very well. Instances are, however, accumulating in alarming number of patients who within one to two months after the reduction cure suffer a complete collapse and develop serious degrees of cardiac insufficiency. The cause for this is, then, usually sought in certain external factors; but in my personal experience the cardiac insufficiency so frequently follows one or another exhausting reduction cure, with the many deprivations that it entails, that an etiologic connection between the two must be assumed. I express the hope that this warning will suffice to prevent obese patients with gout from undertaking exhausting reduction cures in the future.

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